Second Edition

Child Psychology and Psychiatry

Frameworks for Practice

Child Psychology and Psychiatry

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Preface

A key skill that professionals working with vulnerable children need, is to understand how they develop competence. What role do neurobiology and genetic variability play in development? How do relationships with parents and siblings affect social and emotional adjustment? How important is the culture in which families live? We address these questions in the first part of the book. Here, leading researchers and clinicians discuss the physical, social, cognitive and emotional development of the child within his or her familial and cultural context.

Practising clinicians wish to know how to promote children's well-being. In our second section, we address how this can be done throughout the stages of a child's development, including ways of fostering resilience in troubled adolescents. This theme is picked up in subsequent chapters, where authors discuss the clinical and research implications of attachment theory and their influence on children's adjustment to bereavement, adoption and fostering. There are lessons to be learned too, from recent developments in neurobiology and genetics, on individual differences to stress and maltreatment.

Child development does not always follow a smooth or predictable trajectory. Common disorders tend to appear at key developmental stages from infancy through to adolescence.

Feeding disorders usually have their onset in infancy and early childhood. Early intervention is needed to avert persistence into later life; our authors discuss why and how clinicians should intervene. Literacy disorders are an issue for many children at school and can have far-reaching consequences for their educational progress. How to assess and manage this complex learning difficulty is illustrated by our authors. Throughout, the book emphasizes early recognition and recommends interventions that favour optimal outcomes.

Middle childhood is a challenging time as relationships with peers, school and the wider social world become ever more complex. Negotiating one's way successfully requires ever-increasing social awareness and empathy, and an understanding that other people's perspective on things may be quite different to your own. During this developmental period, clinicians see an increasingly wide range of difficulties and disorders emerging. So that we can provide appropriate and effective treatments, it is important to recognize the critical symptoms early. The clinical goal is to build up the resilience of the child and family so that any disruption to the child's psychological, social and emotional development is minimized. In our sections about psychopathology, authors outline the key features of common disorders, alongside guidance on how to intervene. Chapters summarize current key practice points and anticipate future developments.

During adolescence, the child and family must negotiate issues of individuation, autonomy and parental authority. While family support remains crucial to an adolescent's development, their world outside the family exerts a major influence on well-being. Despite the potential for the growth in emotional and social understanding, as well as in increasing independence, this is a time when we find significant internalizing and externalizing psychopathology emerging. Common disorders include depression, self-harm, eating disorders and substance misuse, with some vulnerable young people affected by bipolar or schizophrenic psychoses.

In the latter sections, contributors discuss the specifics of assessment and intervention. What are

the current dilemmas in diagnostic classification? Can the diagnostic systems reflected in ICD-11 and DSM-5 come close to resolving them? When we make a clinical assessment, theoretical frameworks guide us, and these in turn draw on knowledge gleaned from research in neuropsychology, clinical psychology, psychiatry and an understanding of family systems.

Finally, we consider approaches to intervention. When prescribing medication to rectify children's behaviour or emotional adjustment, we will in future be drawing on new findings in pharmacogenomics. Discoveries about the way our genes shape our biology will dramatically influence prescribing practices. While we await such future developments, contributors discuss current best practice in psychopharmacology, as well as reviewing other essential interventions such as parenting programmes, cognitive–behavioural therapy and psychotherapy, alongside systemic and family therapy approaches.

Based upon strong academic foundations, combined with state-of-the art clinical expertise, we provide an essential text not only for trainee clinicians, but also for those wishing to update their clinical practice. Conceived with busy professionals in mind, this introduction to clinical child psychology and psychiatry is concisely written, and its content is clearly presented so as to be rapidly and easily absorbed. Key messages and future directions are highlighted. This book is an essential guide for those working or training in all clinical and community child settings.

David Skuse
Helen Bruce
Linda Dowdney
David A. Mrazek

Section 1

Developing Competencies

Section 1a

Contextual Influences upon Social and Emotional Development

1

Family and Systemic Influences

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Numerous contexts interweave to support children's emotional and behavioural development. In early childhood, family networks are central to those contexts; as children develop, their social worlds expand to take in childcare and school settings, and relationships with friends and peers. Each of these systems, and the interactions within and among them, influences children's growing competencies. Each is also embedded within, and affected by, broader social and cultural influences, and by variations in access to social and material resources. Ecological theories of development [1] emphasize the interplay among these various levels of influence, some proximal to, others more distant from, the child. Figure 1.1 shows a schematic version of a model of this kind, highlighting just some of the broad range of contextual factors known to carry implications for children's emotional and behavioural development.

FAMILY RELATIONSHIPS AND PARENTING

Family relationships are complex: each dyadic relationship is affected by other relationships in the family system, and children both influence and are influenced by those around them [2]. Even very young infants affect the nature of interactions with caregivers, and variations in children's temperamental styles continue to evoke differing responses from carers. In part, variations of this kind reflect children's inherited characteristics; indeed, many aspects of family relationships and functioning once thought to be purely 'environmental' in origin are now known to reflect elements of 'nature' as well as 'nurture' [3]. Children play an active part in shaping the environments they experience; their genetic make-up also affects individual differences in sensitivity to environmental influences, contributing to both resilience and vulnerability to stress [4].

Families are biologically and culturally evolved to promote children's development [5]. Some of the earliest steps in those processes – prenatal and postnatal influences on neurobiological regulation, and early attachment relationships – are discussed in detail in other chapters. But family relationships and parenting show ongoing links with the development of children's behavioural control, and with the regulation of their attentional, arousal and emotional systems throughout childhood. In addition, parents contribute to children's cognitive development; socialize them into culturally appropriate patterns of behaviour; promote their understanding of moral values and the development of their talents; and select and secure their access to key resources beyond the family system.

Successful parenting involves numerous skills and capacities, varying with the age of the child, with culture, and with social context. Underlying this diversity, most models of parenting highlight two central dimensions, one related to parental involvement and responsiveness (encompassing warmth, availability, positive engagement and support), the second centring on 'demandingness' or behavioural control, and incorporating monitoring, expectations and behaviour management. Combinations of these dimensions have been used to characterize four general styles of parenting [6]:

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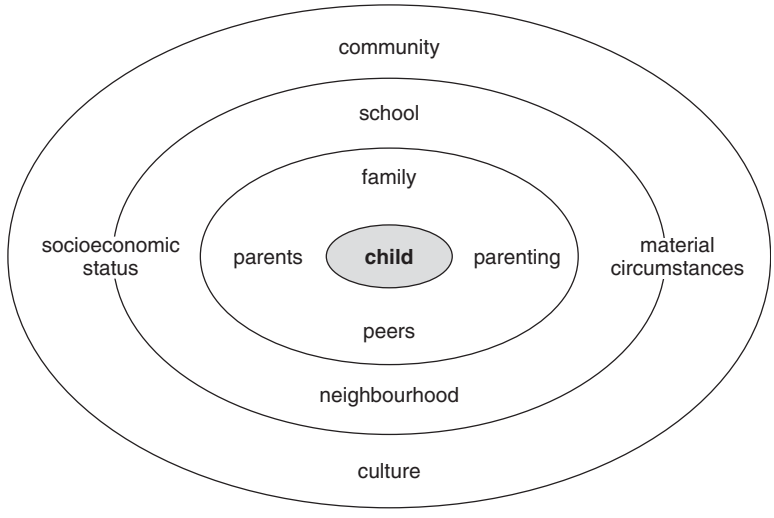

Figure 1.1 Ecological model of influences on development.

- ***Indulgent*** (*responsive but not demanding*): parents are non-traditional and lenient, allow considerable self-regulation, and avoid confrontation.
- ***Authoritarian*** (*demanding but not responsive*): parents are obedience- and status-oriented, and expect orders to be obeyed without explanation.
- ***Authoritative*** (*both demanding and responsive*): parents are assertive, but not intrusive or restrictive. Disciplinary methods are supportive rather than punitive. Children are expected to be assertive as well as socially responsible, self-regulated as well as cooperative.
- ***Uninvolved*** (*both unresponsive and undemanding*): most parenting of this type falls within the normal range, but in extreme cases it might encompass both rejecting–neglecting and neglectful parenting.

Comparisons across these styles consistently highlight *authoritative* parenting as most strongly associated with positive child outcomes in a range of domains: self-discipline, emotional self-control, positive peer relationships and school performance.

When children are under stress, family life can also provide compensatory experiences. Cohesion and warmth within the family, the presence of a good relationship with one parent, close sibling relationships, and effective parental monitoring are all known to represent protective influences of this kind. Finally, when parenting is compromised, risks of emotional and behavioural difficulties increase. The implications of severe problems in parenting, involving abuse or neglect, are discussed elsewhere in this volume, as are family-based risks for specific childhood disorders. At a more general level, risks of this kind appear to reflect problems in four broad aspects of family relationships and parenting:

- Discordant/dysfunctional relationships between parents, or in the family system as a whole.
- Hostile or rejecting parent–child relationships, or those markedly lacking in warmth.
- Harsh or inconsistent discipline.
- Ineffective monitoring and supervision.

Many family-based interventions and parenting programmes are designed to target difficulties of these kinds.

PARENT AND FAMILY CHARACTERISTICS

Some parent and family characteristics also show systematic links with children's risk of emotional and behavioural problems. Parents' own mental health is among the most important of these. In part, these associations may reflect heritable influences; in part, they seem likely to follow from the effects of parents' mental health problems on marital relationships and parenting. Depressed mothers, for example, are known to be less sensitive

and responsive to their infants, and attend less, and respond more negatively, to older children [7]. Alcohol and drug abuse and major mental disorders in parents may impair parenting in more wide-ranging ways; when parents are antisocial, effects may also be mediated through the endorsement of antisocial attitudes and social learning.

Childhood conduct problems are more common among the children of very young mothers [8], often reflecting associated educational and social disadvantages and lack of social supports. The specific contributions of fathers' parenting, father absence and relationships with non-resident parents are attracting increasing attention in research [9]. Large family size may be associated with increased risk for delinquency, but has few links with other aspects of children's adjustment. Only children are not typically at any increased psychiatric risk, and share with other first-borns some small advantages in cognitive development. Birth order also appears to have few implications for behavioural adjustment, although youngest children show some increased rates of school refusal.

CHANGING FAMILY PATTERNS

Recent decades have seen major changes in patterns of family formation and stability in many Western societies [10]. Families are formed later, and are smaller, than in the past; fewer parents marry, and more divorce; and many more women now return to work outside the home when their children are young. As a result, more children today experience out of home and other non-parental care in early childhood, and many also face transitions in their family lives: parental separation and divorce are often followed by periods in single-parent households, and subsequently by the establishment of new step families.

Single parents and step families

On average, children in single-parent and step families show somewhat higher levels of emotional and behavioural difficulties than those in stable two-parent homes [11,12]. In general, however, these effects are modest, and there is much variation within as well as between family types; importantly, associations between the quality of mother–child relationships and children's adjustment are similar across family settings. Single-parent and reconstituted families also often face economic pressures, and may lack social and family supports; mothers may also be under greater stress. Once these variations are taken into account, family type per se shows few consistent links with children's adjustment.

Parental separation and divorce

When parents separate, most children show some short-term behavioural or emotional difficulties; in general, these disturbances are not severe. School progress and motivation may also be affected, and longer-term influences have been detected on young people's own patterns of relationship formation and stability later in life [13]. Research suggests that these responses are not simply 'one-off' effects of parental separation; many children experience parental discord before their parents separate, and divorce itself is often followed by a cascade of other changes. Problematic relationships between parents may continue, and parents themselves are likely to be distressed. In addition, many families face marked declines in economic circumstances, and for some children parental separation will be followed by house moves, school changes, and other disruptions to their social networks. Later outcomes for children may be impacted by each and any element of this complex network of change.

CHILDCARE AND SCHOOLING

By the late 1990s almost half of mothers in the UK returned to full or part-time work before their infants were 1 year old. As a result, grandparents play an increasingly important part in many young children's lives [14], and there has been major interest in the impact of non-maternal care on children's development [9]. Research suggests that multiple features of early childcare need to be taken into account in assessing its effects. Higher *quality* childcare (including, e.g., variations in sensitive and responsive caregiving, and cognitive and language stimulation) is associated with benefits in cognitive and language domains, with better early academic skills, and more prosocial behaviours and fewer adjustment difficulties. Especially in the first year of life, a higher *quantity* of childcare (in terms of hours per week in any kind of non-maternal care), is associated with some increased risks of behaviour problems and disobedience. As

in family settings, individual children's sensitivity to non-maternal care will vary; indeed, for some at-risk young children, out-of-home care has been shown to have positive effects on behavioural development.

School life offers further opportunities, demands and challenges. Starting and changing schools are significant events for all children. Although most adapt well, a significant minority of young children show some difficulties when they start school, and many young adolescents show short-term declines in both their academic performance and their self-esteem when they transfer from primary to secondary school. Tests and examinations figure high on children's lists of fears, and major examinations are often associated with some increases in psychological distress. Bullying – a problem especially associated with the school context – is attracting increasing attention as a risk factor for children's mental health [15]. Surveys suggest that quite large proportions of children experience occasional bullying at school, and that smaller groups are persistently victimized. Although such children may have shown anxious and insecure behaviours before they started school, there is now clear evidence that bullying has independent effects on risks of later adjustment problems.

Like families, schools vary in their social and organizational 'climates' in ways that have modest but independent effects on children's academic progress and behaviour [16]. In part, these variations reflect variations in the background characteristics of the children each school admits; in part, they seem attributable to differences in organizational characteristics and the tenor of day-to-day school life. Schools with more positive child outcomes have consistently been found to be characterized by purposeful leadership, constructive classroom management, an appropriate academic emphasis, and consistent but not oversevere sanctions. For behavioural outcomes, the composition of pupil groupings may also be important. Young children are more likely to become aggressive if they are placed in classes with other very aggressive children, and risks of delinquency may be increased in secondary schools with large proportions of low achievers. By the same token, school- and classroom-based interventions can prove highly effective in behaviour management, and for some severely disadvantaged children schooling can be an important source of positive experiences and support. In addition, experimental studies of preschool programmes have documented important long-term gains in terms of reduced risks of delinquency and unemployment many years after participants left school.

WIDER SOCIAL AND ENVIRONMENTAL INFLUENCES

Poverty and social disadvantage

Poverty and social disadvantage are consistently associated with variations in children's health, cognitive skills and academic achievements, and – though somewhat more modestly – with their social and emotional development [17]. Disruptive behaviours in particular show links with persistent family poverty, with effects that are more marked for boys than for girls, and are stronger in childhood than in adolescence. Research suggests that these associations reflect elements of both social selection and causal influences. Especially in families of young children, effects are likely to be indirect, operating through processes whereby poverty imposes stresses on parents, and these in turn impact on family relationships and parenting [18]. In more affluent societies, relative deprivation – the perception of disadvantage by comparison with others – may also contribute to parental stress.

Neighbourhood and community contexts

Rates of behavioural difficulties (and other markers of child health status) also vary with neighbourhood context [19]; problem levels may be especially high in chronically disadvantaged inner-city areas, and the task of parenting may be more challenging when neighbourhood supports are poor. Once again, many of these effects seem likely to be indirect in early childhood, operating via increased stress on families. But in severely disadvantaged settings even quite young children may be directly exposed to community violence, and later in development neighbourhood influences may be mediated through associations with delinquent peers.

Multiple stressors

For many children, exposure to these and other adversities will covary: children in stressed families

may also live in poor neighbourhoods, attend poorly resourced schools, and be exposed to deviant peers. Research suggests that risks at the child, parenting, peer and sociocultural levels each add uniquely to the prediction of emotional and behaviour problems. The total number of risks explains further variance in outcomes, and evidence is beginning to accumulate that differing *configurations* of risk are associated with specific emotional and behavioural difficulties [20]. Exposure to poverty, for example, may differ in its impact depending on parental characteristics and the quality of family relationships; comprehensive assessments of family and systemic influences require that each of these levels of influence, and the interplay between them, be taken into account.

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2

Sibling Influences

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WHAT'S NEW

- Sibling relationships are characterized by strong emotions: positive, negative and ambivalent. Coupled with the intimacy of their relationships (siblings know each other very well from early infancy onwards) this emotional intensity means the potential for developmental influence is large.
- Longitudinal research has documented connections between friendly early sibling relationships and children's later social understanding, prosocial behaviour and adaptive functioning. Evidence for continuities in quality of sibling relationships is accumulating.
- Conflict between siblings is now regarded as a normative feature of sibling relationships. The impact of parents on sibling conflict is currently under scrutiny, while sibling bullying at home has been shown to relate to the experience of being bullied at school. A warm sibling relationship has a protective effect on children growing up in families with a high level of marital conflict, or faced with negative life events.
- Siblings growing up in the same family differ notably in adjustment, in personality and well-being; this is a major challenge to understanding family influence. Processes implicated include differential parental treatment, and other 'non-shared' experiences within and outside the family.
- Children's perspectives on their sibling relationships are increasingly recognized as important, and can be reliably studied.
- Interventions have focused on reducing sibling conflict; a new direction is to promote the positive features of sibling relationships.

The great majority of us grow up with siblings, and the sibling relationship is the longest-lasting we are likely to experience. How important are siblings as an influence on the way we develop? Clinicians and family therapists have long argued that siblings play an important and influential part in children's development, but until the last two decades, systematic research on sibling influence was relatively sparse. What has recent research told us about the factors that affect the ways in which siblings relate to one another? Is there continuity over time in the friendliness or hostility between siblings? What is the evidence for sibling influence on adjustment and well-being, on social and emotional understanding, and on children's other relationships?

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INDIVIDUAL DIFFERENCES IN SIBLING RELATIONSHIPS

From infancy to adolescence, relationships between siblings are emotionally powerful [1]. Observational studies report that for some siblings, the majority of interactions between siblings are intensely negative, for others positive emotions are frequently expressed, for others the emotional quality is ambivalent. Continuities in the emotional quality of the relationship are evident from preschool years through middle childhood [2].

Why do siblings differ in their relationships with one another? Research in the 1970s and 1980s focused chiefly on birth order, gender and age gap as sources of individual differences. For young children the evidence on the importance of age gap and gender for sibling relations is inconsistent; in middle childhood gender differences become more apparent, with boys reporting less warmth and intimacy with their siblings. Links between children's temperamental characteristics and their relationships with their siblings have been reported, but findings are inconsistent across studies [3]. Recent research on siblings has now broadened to include the quality of close relationships within and outside the family as sources of individual differences [4], and the evidence for sibling influence on children's sociocognitive development.

SIBLING RELATIONSHIPS AND PARENT–CHILD RELATIONSHIPS

Positive relationships with parents are linked to friendly, caring relationships between siblings, while negative relationships between parents and children are associated with sibling hostility. Children who have secure attachment relationships with their parents are reported to have positive relations with their siblings [5]. But causal conclusions cannot be drawn from these associations: while such links are often interpreted as evidence for parental influence, it could well be that children's temperamental qualities contribute to difficulties in relationships with both sibling and parent. While a sunny, easy-going child's temperament may contribute to positive relationships with both parents and siblings, constant quarrelling between siblings may contribute to difficult parent–child relationships, and indeed to difficulties in the relations between parents.

In contrast to this evidence for hostility across family relationships, some studies report that supportive sibling relations can develop in families in which parent–child relations are distant or uninterested [6]. These 'compensatory' patterns of family relationships may be more common in families facing stress and social adversity. Siblings can also be sources of support for children growing up in homes with marital conflict, and longitudinal research shows that children have fewer adjustment problems following negative life events if they have a good warm relationship with a sibling (Figure 2.1) [7].

A further point about the complex patterns of links between relationships within the family concerns the consistent evidence that in families in which there are *differential* relations between parents and their various children – where more affection and attention, or more negativity or harsh discipline is shown towards one sibling than to another – there is more hostility and conflict between the siblings [8]. These links are particularly clear in families that are under stress. Causal inferences cannot be made, however, if the studies are cross-sectional. Recent evidence has shown that children's interpretation of differential parental behaviour is important [9]. When children interpret their parents' differential behaviour as evidence that they are less worthy of parental love than their siblings, the sibling relationship is particularly likely to be compromised [10]. These findings remind us how important it is to recognize the context of multiple family relationships within which siblings grow up. From the second year on, children monitor the interactions between their parents and siblings with vigilance [11].

The evidence that influential experiences within the family differ markedly between siblings has shown us that it is sibling-specific experiences that need to be studied [4]. Innovative analytic techniques have been developed to assess and distinguish between these 'child-specific' and 'family-wide' influences [12].

SIBLINGS AND THE DEVELOPMENT OF SOCIAL UNDERSTANDING

A striking feature of sibling relationships is their intimacy. Siblings know each other very well. When young they spend more time interacting with their siblings than with their parents or friends, and

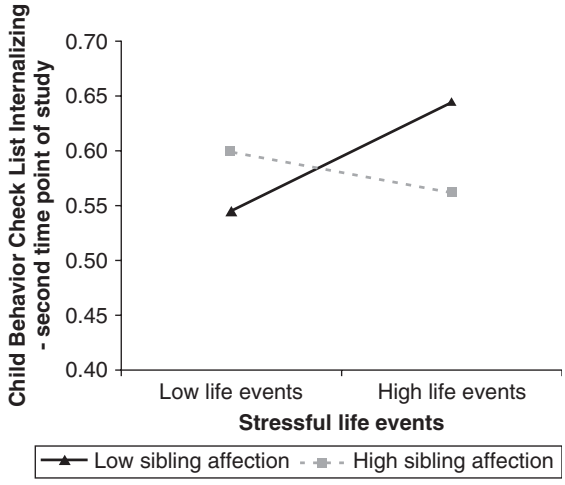

Figure 2.1 Internalizing symptomatology at second time point as a function of stressful life events and sibling affection at first time point. Reproduced from Gass *et al*. [7], with permission.

from very early in childhood know how to upset, tease and irritate their siblings as well as how to comfort and amuse each other. Sibling research has given us an important new perspective on a key aspect of cognitive development – children's discovery of the mind. The growth of children's understanding of emotions, thoughts and beliefs, and the links between these inner states and people's behaviour is a core feature of early cognitive development, and one in which sibling relations can play an important part. Children with siblings begin to show powers of anticipating others' intentions, sharing an imaginative world [1], and they engage in talk about why people behave the way they do. Individual differences in experiences of conversations with a sibling about feelings and inner states, and cooperation in shared pretend play, are linked to differences in children's developing maturity in social understanding. While the issue of direction of influence remains a difficult one, the study of siblings has highlighted the key social processes within the family (e.g. sharing cooperative pretend play, and managing conflict) for these core developments in social understanding. The key distinction is not between only children and those with siblings, but rather the individual differences in the quality of the relationship between the siblings. It is a warm, affectionate sibling relationship that is linked to the growth of social understanding.

SIBLING INFLUENCES ON ADJUSTMENT

Evidence for links between children's relationships with their siblings and their aggressive oppositional behaviour, and also their internalizing (worrying, anxious and depressive behaviour) has accumulated [1,13,14]. The influence is both from older to younger siblings and vice versa. Low levels of prosocial behaviour (caring, empathetic and helpful, supportive behaviour) are associated with hostility between siblings and the development of conduct problems [14]. These patterns are independent of the contribution of poor parent–child relationships, and are evidence for *direct* effects of sibling conflict and negativity on children. *Indirect* effects of siblings on adjustment have also been found, for instance in the impact of differential parent–child relations on children's adjustment problems [15].

The evidence from research on the very beginnings of the sibling relationship – the impact of the birth of a sibling on children's well-being – is also clear. Increases in problems of aggression, dependency, anxiety and withdrawal have been reported for first-born children following the birth of a sibling [1].

SIBLINGS AND PEERS

Research that includes children's perceptions of their relationships with siblings, friends and

mothers, and links with their adjustment paints a complex picture. This can be illustrated by Stocker's study of the self-reports of 7–8-year-olds on their relationships [16]. First, warmth in relations with each partner was associated with fewer feelings of loneliness in peer contexts and better behavioural conduct. Warmth in sibling relationships and in friendships was associated with a sense of self-worth, and in friendship was linked to less depressive mood. There was evidence for some correlations across the different relationships, but these correlations were not high. Compensatory processes were found, for instance, in children's sibling relationships and friendships in relation to behavioural adjustment. High levels of warmth in friendships compensated for low levels of warmth in sibling relations (and vice versa) in relation to adjustment outcomes. Finally, children who perceived low levels of warmth in their relationships with both mother and sib had significantly worse outcomes on loneliness, self-worth and behavioural conduct.

Bullying victimization by siblings at home has been reported, and linked to bullying at school and to adjustment problems [17]. In a study of 12–15-year-olds in Israel, 16.2% were bullied at home by siblings, and more than half of these were also involved in bullying incidents at school. Ethnic (Jewish vs Arab) and sex differences were small compared to the effects of the sibling relationship on behaviour problems. The findings indicate that intervention programmes targeted at children's close friendships and sibling relationships may be important in improving children's well-being.

INTERVENTION PROGRAMMES AND SIBLING RELATIONSHIPS

The frequent conflicts between siblings, the evidence for sibling bullying and for links between sibling disputes and children's aggressive behaviour has led to an emphasis on reducing sibling conflict as the key mechanism for improving the relationship [18], for example by training parents [19]. The short-term effects of parental mediation (encouraging reasoning, discussion of emotions, and taking the perspective of the other child) were studied with 5–8-year-olds in Canada. Children responded appropriately to the mediation, and the programme empowered children to solve conflict issues. These programmes do lead to reduced conflict, but fail to increase the positive aspects of the relationship. In contrast, Kramer has set out a useful review of the positive aspects of the relationship, and devised a programme for intervention with siblings and parents, 'Fun with Sisters and Brothers', based on the competencies highlighted in the review [20]. These competencies include:

- play;
- conversation;
- mutual enjoyment;
- valuing help and support;
- appreciating sibs' unique knowledge of each other;
- learning to respect sibs' views and interests in addition to one's own;
- managing emotions in challenging situations;
- learning to check faulty hostile attributions;
- refraining from wild behaviour or bossiness;
- conflict management; and
- for parents, discussing the impact of parental differential treatment.

The contribution of the programme to siblings' relationships is awaited with great interest.

It should be noted that most studies of siblings have been conducted with middle-class, urban, two-child, Caucasian families in North America or Britain; little is known about minority ethnic or linguistic groups. There are clear and important opportunities for research, using longitudinal studies, to fill some of the gaps in what we know about this interesting, intense and life-long relationship between siblings.

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3

Culture and Child Development

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INTRODUCTION

With increasing contact between cultural and ethnic groups the concepts of childhood and the child itself have become sites for intensive study. Anthropologists, historians and cultural child psychologists have attempted to study children's lives and conceptions about childhood in different cultural contexts. Wide differences exist across cultures [1]. For professionals working in pluricultural contexts a pertinent question is: how are ethnic differences accounted for in prevailing concepts of parenting and childhood behaviours, both 'normal' and 'variant'? These complexities are amplified by the continually changing social and political contexts in which children grow up, and highlight the very social and historical framing of constructions of childhood. Ethnic variations in parenting may express adaptations to different conditions for child development necessitated by divergent ecological, social and cultural priorities, rather than inherent, static 'ethnic differences'.

DEVELOPMENTAL NICHE AND ECOCULTURAL PATHWAYS

The concept of a developmental niche was introduced as a framework for studying the production of health and development of the child via the interaction between the physical and social settings of the child's everyday life, culturally determined customs of childcare and parental theories about children [2]. The components of the niche form the larger strategy for childcare and the daily routines for the child. The everyday routines embody the core goals and fashion cultural developmental pathways for children in specific ecocultural contexts [3]. LeVine [4] demonstrates how in parts of the world where child survival is precarious, close physical proximity with the baby is maintained by carrying, co-sleeping, breastfeeding, immediate response to crying, and substitute care by siblings when the mother is working. The pursuit of learning is postponed until survival is assured. The increased physical contact and stimulation promotes growth, and development of attentional processes and neuromuscular competence [5]. By contrast, in technologically advanced North America, where child survival is of less concern, but preparation for competency in future occupational roles graded by mastery of literacy-based skills is important, mothers emphasize the attainment of language skills and mastery of the object world through communicative interaction and naming of objects from an early age, rather than close physical proximity.

The models are also useful in understanding changes in parenting styles related to alterations in the ecocultural context.

CHILDHOOD AND PARENTING ACROSS CULTURES

As most accounts of children's development and needs are framed within writings from North America and Europe, the normative description of childhood is often based on children growing up within the Northern cultures, and even within this

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differences across class and socioeconomic strata are subsumed. Culture is often consigned to the role of an 'add on' variable to assumed normative standards applied universally. However, recent cultural studies of children have questioned assumptions about 'cultural universals', and have highlighted the centrality of culture in shaping human behaviours, crucially here of parents and children. How integral is culture to concepts regarding childhood, child development and the goals that frame parental strategies for bringing up children? Although children achieve developmental maturity along broadly similar species-specific lines, and the goal of parenting is similar across cultures, namely for children to become competent adults in one's own cultural, moral and economic world, there are wide differences in what constitute the desired competencies and the means for achieving them. Shweder [6] makes the point that any aspect of human nature that we endeavour to understand must have a central essence, but that essence consists of a heterogeneous collection of structures and inclinations substantiated by the historical experiences of different cultural communities, resulting in 'One mind, many mentalities: universalism without uniformity'. This is different from culture being perceived as variations from a normative standard. For children, James [7] eloquently posed the question: 'One childhood or many?' These issues are further explored via examples commonly encountered in cross-cultural work.

INFANCY

Developmental stages

The very notion of stages of childhood is culturally constructed. Developmental psychologists mark the end of infancy with the beginning of 'toddlerhood' normatively defined at age 2 years and marked by language and motor competency. However, this is not a biological fact, but a cultural convention based on the assumption that life stages should be delineated by absolute points in time. It is different from norms in cultures where the crucial reference point is the acquisition of moral sense [8]. Puritans of New England began strict discipline at age 1 year, when they believed infancy ended and the Devil begins to exert control. The ethnographic record shows that in most parts of the world active teaching begins at least after 5 years, as it is believed that before this children are too immature or lacking in 'sense' to be taught important lessons [1]. The Baganda of Uganda typically train their infants to sit independently as early as 4 months as sitting up and smiling is an asset amongst the Baganda, who value highly face-to-face contact [8].

Attachment

Cross-cultural research on attachment behaviour has thrown up challenging questions regarding how deviation from supposed universal norms is to be understood at a population level. The Bowlby–Ainsworth model of attachment describes a universal model of attachment behaviour predicated on the primary carer's sensitivity to the infant's signals. Ainsworth's work in different parts of the world testifies to the universality of attachment behaviour in infants, with group B attachment behaviour being modal in most cultures [9]. However, the model does not allow for variations other than as suboptimal or pathological. A review of the literature on attachment taking into account population variations, raises the question whether there is a multiplicity of optimum patterns for humans [10] reflecting different meanings attributed to optimal patterns of attachment behaviour in different sociocultural groups [9]. For example, a study of attachment [11] in Bielefeld (Germany), showed 49% of the infants to have anxious-avoidant attachment behaviour, which related to a highly valued cultural emphasis on obedience and self-reliance, the training for which began in infancy. It was an accepted practice to leave infants in bed alone for short periods, and mothers compared the extent to which their babies could play alone as indication of their developing self-reliance. Given the absence of evidence for a higher prevalence of personality disorder in Bielefeld, it could be argued that the drive for self-sufficiency engendered behaviours that counted as virtuous in some German communities, and delineates a different pathway towards normal emotional development. Historically, the prevention of infant dependency was also highly valued amongst British and American middle classes until the end of World War II.

The variety in infant care-taking patterns is highlighted by hunter gathering Efe (Zaire), where multiple care-takers alongside the mother, provide

both lactating and non-lactating care, resulting in the Efe infant being more diffusely attached to many caregivers rather than intensely attached to one. Tronick *et al.* [12] propose that this model of care-taking is moulded by specific ecological demands and cultural values that emphasize group identification.

Co-sleeping

Co-sleeping is widely practised in many parts of the world such as Africa, Asia and the indigenous Americas, particularly where the economic unit of production was until recently the family. Babies and young children sleep with parents, grandparents or older siblings even where room is available. Co-sleeping promotes togetherness in cultures where the family is the economic unit and interdependency highly valued. In advanced industrialized Northern economies where autonomy and independence are emphasized, babies sleep apart from parents. Neither practice is morally superior but each is tied to the competency required for the specific cultural context.

Parental involvement in play and learning

Parental facilitation of the child's academic preparedness through proto-conversations with babies, active teaching through toys and make believe is considered critical by middle-class Euro-American parents as it promotes skills required for future school and occupational success. Although the parent as teacher is often enshrined as an ideal in manuals for parents, it is essentially a cultural model that promotes important developmental skills required in complex urban societies. It is not an ideal in agrarian pastoral societies where processes are less demanding and where everyone is a potential teacher or role model. Instead qualities such as initiative, attention to details, sharing, obedience and respect for elders attract greater value.

MIDDLE CHILDHOOD

With the introduction of compulsory education in most parts of the world, middle childhood has received relatively less attention in cultural work. However, a closer examination reveals startling differences in how children's lives are structured at this stage. Ethnographic description of the Girima (Kenya) encapsulates some differences from the North American normative expectations of children at this stage, but ones that are also seen in many other cultural groups in the developing world [13]. Girima attach importance to providing children with duties that teach responsibility and mutuality necessary for future adult cooperative roles. Children aged 2–3 years take pride in running errands, and from 8 years a girl may be expected to pound maize and a boy to herd. Work constitutes opportunities for acquiring skills in future gender-specific roles, as well as opportunities to participate in cooperative activities with other children. These activities are often combined with attending school. Assistance within the home is different from wage labour, which, however, remains a reality for children from socioeconomically very deprived families in many parts of the world, and which keeps the world price of commodities down [14].

ADOLESCENCE

Adolescence as a stage between childhood and adulthood where the participants behave and are regarded differently appears to exist worldwide. This stage may therefore not be a product of culture, although many of its descriptions are [15]. In many cultures where socialization into adult occupational roles begins early, it is less, as described in the West, a stage when identity questions about future roles begin, but rather a stage for preparation for future reproductive roles, within which individuation is subsumed. The emphasis at this stage on individuation and identity formation that is adaptive for industrial and ever-changing capitalist economies, is not shared across cultures where the social-relational self is emphasized.

ETHNICITY AND MENTAL HEALTH

This section addresses the relationship between ethnicity and mental health problems. However, before a cultural explanation is offered to explain differences between ethnic groups, important methodological considerations need to be taken into account, particularly whether 'like is being compared with like' in terms of sampling, socioeconomic variables, ethnic groupings, population versus clinic rates, cross-cultural validation

of instruments and diagnostic categories, and accessibility of services.

Cross-cultural epidemiological studies are too varied for firm conclusions to be drawn about worldwide rates and patterns. There is little evidence for culture-specific syndromes, but dissociative disorders such as trance and possession in adolescence related to rapid social change in parts of the world where possession beliefs exist, are reported [16].

The systematic review of the mental health of children of the main ethnic groups in Britain by Goodman *et al*. [17] shows comparable, if not better mental health in minority children, given the socioeconomic deprivation of Pakistanis, Bangladeshis and Afro-Caribbeans in Britain, and it underscores the need to research the interplay between risk and protective factors for different communities. A few child psychiatry disorders will be used to explore the complexities involved.

A systematic review of attention deficit hyperactivity disorder [18] estimated the worldwide pooled prevalence at 5.29%, although notably approximately two-thirds of the 102 studies were from North America and Europe. The authors emphasize caution in interpreting the results because of the variability in findings. Variation in prevalence ranged from 1% to 20%. Despite attempts at standardization, significant differences between raters from different countries remain. This has provoked a debate about cultural constructions in diagnosis and treatment, and whether the differences in ADHD rates also reflect different thresholds in tolerance for non-conforming behaviours in children [19].

The evidence for the role of culture in the aetiology of eating disorders related to weight consciousness is evidenced by wide variation in rates worldwide, and rising rates attributed to culture change through urbanization and modernization. In The Netherlands, Van Son [20] found a fivefold increase of bulimia with urbanization between 1985 and 1999.

A review of non-fatal self-harm in the UK [21] described higher rates amongst South Asian female adolescents. Notably, a high prevalence is not reported from South Asia. Although the authors refer to culture conflict, other explanations need to be considered. For example, Reese [22] found that migrant parents who perceive a higher risk for young people in the new environment exercise greater boundary control over adolescents than would be exercised in the country of origin, resulting in greater inter-generational conflict. Self-harming behaviour itself may be an appropriated cultural way of expressing distress by South Asian girls in the UK.

CONCLUSION

As culture and ethnicities are always evolving, a foreclosure of the debate is never possible. A more productive alternative is the development of a framework for understanding the centrality of culture in child development based on the extensive cross-cultural literature available. However, as familiarity with one's own cultural norms is often the starting point for studying difference, 'cultural difference' is often subsumed by the issue of 'different moralities'. But for mental health professions culture is a potent tool for promoting reflexivity, and widening our horizons by including knowledge about the everyday lives of children from parts of the world where the majority of children live.

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4

Neurobehavioural Development in Infancy

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WHAT'S NEW

Neurobehavioural development is not simply a biological process, but one that is shaped by the regulation that takes place through interactions between caregiver and infant. Notably these interactions are not simply 'biologically determined', but vary by community and even by dyad. Understanding the infant neurobehavioral state is a significant approach to assessing their capacities. It is also one way to understand the effects of adverse biological and social factors on their neurodevelopment.

Infant neurobehaviour and its development include behaviours generated by neurophysiological and psychological processes, which mediate infants' own internal processes and engagement with the world. Ideally, neurobehaviour becomes adaptive within contexts that challenge infants on a daily basis. The purpose of this chapter is to introduce theory-driven and empirically supported influences on neurobehavioural development while emphasizing mechanisms currently known to exemplify the interaction of neurobiological and social domains of development. We begin with a rather narrow overview of neurobehaviour, followed by the argument that a broader biosocial view is needed to understand neurobehavioural development. Although it may be surprising to some readers, we argue that neurobehaviour is not self-contained, nor preset, nor a simple unfolding under the control of genetic maturational processes. We see neurobehaviour as embedded in regulatory processes between infants and caregivers that operate in a continuous, bidirectional and dynamic manner. Furthermore, these regulatory processes are affected by multiple factors from health status to toxic exposures, not the least of which is culture. We see these regulatory processes sculpted by culture, in addition to biological and physiological processes. This broader perspective implies that neurobehaviour affects and is shaped by regulatory processes including the psychosocial and biological contexts that make up human experience.

INFANT NEUROBEHAVIOURAL CAPACITIES

In the past, newborns were seen as reflexive. Infant neurobehaviour was modelled on spinal frog behaviour, where responses to stimuli were thought to be fixed, under stimulus control, and automatic; but the model was dramatically wrong [1]. The newborn's brain and physiology produce four domains of complex neurobehaviour:

1. *attention* – visual and auditory abilities to process information such as tracking objects, discriminating faces;

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2. *arousal* – the expression and intensity of states from sleep to alert to distress and their modulation using self-soothing of distress;
3. *action* – fine and gross motor skills for acting on the world of things and people such as defensive 'reflexes', reaching for an object;
4. *affective social processes* – communicative emotional displays, smiling [2].

However, understanding infant neurobehaviour requires a recognition that the quality of neurobehaviour is mediated by state – the organization of neurophysiological (heart rate, respiration, EEG) and behavioural systems (tone, movements) for a period of time. Six states have been identified and are now used to understand infant neurobehaviour: two *sleep* states (State 1, quiet sleep; and State 2, REM sleep), one *semi-awake/transitional* state (State 3), two *awake or alert* states (State 4, quiet alert; and State 5, active alert) and a *distress* state (State 6) [3,4]. Recognizing the mediating role of state overturned the spinal frog model when infant reflexes (e.g. knee jerk, sucking) were found to vary in intensity, robustness and quality depending on the infant's state [5]; that is, they were hardly single synapse spinal reflexes.

Beyond its effect on reflexes, each state impacts the quality of infant neurobehaviour. States affect the infants' repertoire of complex motor and sensory/perceptual processes [3,6] and even determine various infant response modalities; for example, facial brightening and alerting to visual stimuli only occur during the awake states; startles occur in States 1, 4, 5 and 6, but seldom in State 2 or 3; movements are smooth in State 4 but jerky and uncoordinated in State 6, uncoordinated in State 3 and largely absent in State 2 [7]. Furthermore, infants collect information and modulate their behaviour differently in different states. Head turning to sound and cuddling occur primarily in States 4 and 5, may occur in State 3 but not in States 2, and habituation can occur in States 1, 2 and 4.

Infant neurobehaviour can be evaluated. The NICU (Neonatal Intensive Care Unit) Network Neurobehavioral Scale (NNNS) is one such neurobehavioural measure. As a standardized assessment tool for infants [8,9], the NNNS assesses infants from the newborn period to later in the postnatal period, as well as pre- and post-term at-risk infants (e.g. drug-exposed, jaundiced). Using different stimuli (bell, rattle, ball, human face and voice) and handling techniques (cuddling, stimulation of neurological reflexes), the NNNS elicits a variety of attentional, motoric and regulatory responses and capacities to *inhibit* responses to insignificant stimuli. Critically, the NNNS considers infant state for each neurobehaviour and tracks the range of states and their lability. The NNNS gives a holistic picture of the infant by assessing the interplay of state behavioural and regulatory capacities.

The NNNS is sensitive to risk factors that affect infant neurobehaviour [10,11], such as gestational age, birthweight, appropriateness of growth, postnatal age at testing, quality of care and stress reduction of different delivery procedures, *in utero* exposure to drugs (cocaine, heroin, methadone, nicotine), and also maternal stress and depression [12]. Impressively, NNNS profiles of infants' neurobehavioural organization ('well organized' to 'poorly organized') have predicted long-term outcomes related to school readiness and IQ at 4.5 years of age [11], which speaks to the significance of infants' self-organized neurobehavioural capacities for their long-term psychosocial development.

MUTUAL REGULATION MODEL

Although we see infants as competent beings with an impressive ability to self-regulate and to act upon the world, we must recognize that their capacities are limited and immature. The quality of neurobehaviour dissipates unless infants receive external support to scaffold their organization. For instance, infants are able to control heat loss by taking a fetal position or increasing their activity, but may require caregiver ventral contact to achieve thermal homeostasis. Similarly, infants are capable of processing stimuli in an alert state, but that state is energetically costly and often short lived. They are better able to sustain neurobehavioural alert states with postural support and soothing from their caregivers [13]. Altogether, infants actively regulate their neurobehavioural systems to maintain homeostasis and neurobehavioural coherence, especially when supported by a caregiver. Such external supports are often overlooked, but are critical to the quality of expression and development of infant neurobehaviour.

We propose the use of the Mutual Regulation Model (MRM) to describe external supports that lead to organized infant neurobehaviour. It has

been established that infants and adults jointly regulate their behaviour, affect and communication during social interactions, and share goals to attain well-organized physiological, behavioural and interpersonal states [14,15]. The MRM proposes that these processes are jointly regulated by infants using internal self-organized capacities in coordination with regulatory input from caregivers [16,17]. The success or failure of mutual regulation to maintain infants' neurobehavioural coherence and engagement with others thus depends on both infant and caregiver capacities: the integrity of the infant's physiological and central nervous systems, the infant's abilities to express the status of these systems to the caregiver, and the caregiver's abilities to read and act upon the infant's messages. These processes are dynamic such that each continuously influences and modifies the other; they are mutually regulated.

CULTURE

Once infant neurobehaviour is considered deeply interdependent with the actions of another person, a broader view of neurobehavioural organization is required. It needs to be considered as a socially regulated process, not simply a biological process. As a social process, the brain and physiological processes controlling neurobehaviour are imbued with culture [18]. This perspective is demanded because culture determines caregivers' implicit and explicit view of infant capacities and their attention and actions with their infant. This is seen in the way that the caregiver responds to the infant's needs moment-to-moment. Thus caregiving practices within cultures influence the development of infants' central nervous systems and infants' responses to the particular constraints set by the cultural environment.

Yet, the role of culture on neurobehavioural development has traditionally been overshadowed by biological factors, maturation and the effects of biological perturbations (e.g. illness, malnutrition) in part because they often demonstrate causal and immediate effects on physiological and behavioural changes; the presupposition is that these factors are the primary determinants of neurobehavioural development. The MRM, on the other hand, posits that prenatal and postnatal experiences actually sculpt functional and structural characteristics in neurodevelopment, making culture much more proximal to neurobehavioural development. Although studying cultural effects is difficult compared to factors that can be manipulated (e.g. NICU care) or that already vary within a community (e.g. exposure to toxicants), characterizations of infant development from various cultural groups support the interdependence of biology and culture, as does research on gene–environment interaction and epigenetic processes [13,19].

For instance, unique care-taking practices among the Peruvian Quechua influence the biological and behavioural processes underlying infant neurobehavioural development. Peruvian Quechua dwelling at high altitude (4250 m) use a culturally created care-taking technology, the Manta pouch, to 'house' their infants – a layered set of cloths and blankets that tightly wrap around and fully enclose the infant. It protects infants from the extremes of the environment (i.e. freezing temperatures, reduced oxygen, lack of humidity) [20]. Within the Manta pouch, the temperature is stabilized and high, the air is humidified, and infant movement is limited. Paradoxically, the O_2 levels are lower and CO_2 levels are higher in the pouch compared to hypoxic conditions at high altitude. While these hypoxic conditions would be considered dangerous in other environments, the increase of CO_2 may actually be a microstressor that induces adaptive functional and structural changes. In combination with other features of the pouch microclimate, high CO_2 levels increase the duration of infant sleep, which conserves energy; this in turn leads to faster physical growth and resistance to temperature loss. Raised CO_2 levels are partly responsible for inducing the left shift of the Quechua's CO_2 sensor, a unique physiological characteristic that allows them to tolerate high levels of CO_2 that would debilitate people lacking this adaptation. The left shift is a developmental phenomenon directly related to the length of time an individual lives at high altitude prior to puberty.

Although adaptive, the Manta pouch may be costly to other aspects of neurobehavioural development. Quechua infants are immobile and completely covered around the face while being carried on their mothers' backs during infancy. Stimulation and social interaction are limited; the limitations in socialization and stimulation may reduce exploration and language development

and other features of brain development. In fact, the infants have been found to lag developmentally. Thus fundamental physiological processes – tolerance for CO_2 – and fundamental neurobehavioural processes – amount of sleep, motor development, and perhaps the development of emotions such as curiosity – are affected by Quechua care-taking practices. Nonetheless, the protective shielding may be important for working at high altitude and one can speculate that the Manta's experience of isolation perhaps prepares the 6-year-old Quechua child to endure the social isolation and vigilance needed for herding their family's animals alone for days at a time.

Such an example may seem extreme to Western societies, but is not farfetched in light of challenges that practitioners face when working with families that hold different cultural expectations for infant development and preferences for caregiving. Currently, Western practitioners urge caregivers to place their infants on their back for sleep. After noticing motor delays (e.g. turning, crawling) as a result of this change in sleep position, many practitioners consider it essential for caregivers to practice 'Tummy Time' with their infant – 30 minutes of daily exercise where the infant strengthens neck muscles in preparation for sitting up and crawling [21]. Furthermore, caregivers are given recommendations in the way to socially engage their infant during this time [22]. Perhaps this recommendation emerged from the recognition that an infant with poor head control is less able to attend to the environment and to engage in affectively charged face-to-face exchanges with caregivers, a norm in the United States. By modifying infant neurobehaviour, motor and social development is now viewed again as 'normal'.

However, 'Tummy Time' may not be readily adopted by caregivers from certain cultures; for instance, Asians have always placed their infants on their back to sleep and never considered infant motor milestones delayed. In sub-Saharan Africa, infants are not played with in the same way as infants in the West and recommendations to do so would not be accepted. In fact, motor development is *accelerated* compared to the motor development of infants in the West from being carried from birth [23]. The neurobehavioural development of infants who experience 'Tummy Time' may differ from those who do not, yet each may be the preferred development within their respective culture. As decisions made by caregivers who do not readily adopt Western recommendations may be considered problematic by Westerners from a biopsychological perspective, practitioners ought to evaluate how these caregiver decisions fit with the developmental goals of their own culture. Cultural comparisons indicate that there are no fixed and universal norms.

CONCLUSION

The consideration of only biological factors in neurobehavioural development is an insufficient view of neurobehavioural development. A broader view of neurobehavioural development is necessary to capture the complexity of the processes that unfold over time. Infant state acknowledges the overall organization of infants' physiological and behavioural systems as it receives external inputs from others. Through the MRM perspective, caregiver and infant jointly make efforts to maintain infant neurobehavioural coherence and organization. Moment-to-moment organization of neurobehaviour is partly governed by social input. Over the long run, the interplay of self-organized neurobehavioural processes, caregiver practices, and resulting interpersonal states – however they may differ across cultures – organizes infants in a coherent and culturally appropriate manner. And most telling, infants who are unable to engage socially in an appropriate way within their culture will not develop normally. Fortunately, good parenting naturally flows when caregivers function well in their environments, which in turn helps infants to develop into culturally appropriate beings.

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5

Genetic and Biological Influences

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INTRODUCTION

The Human Genome Project was completed in 2003 [1], and the first draft sequence of DNA in a human cell was then available for everyone to scrutinize. What have we learned since, and have those insights benefited patients with psychiatric disorders?

Until relatively recently, what little was known about genes that contribute to neurodevelopmental conditions came from the study of single gene mutations, in which the normal activity of an individual gene is disrupted. Disorders attributable to errors in a single gene are exceptionally rare (typically with an incidence of no more than 1 per 10 000). Remarkable advances are being made in our understanding of the pathogenesis of 'single gene' conditions such as Fragile X or Rett syndrome, and new opportunities for treatment are being discovered that could potentially have wider applications [2,3].

Unlike single gene disorders, most psychiatric conditions are highly complex in terms of their underlying genetic predisposition. Whilst twin and adoption studies indicate an important role for heredity, risk is attributable to the sum of individual differences in hundreds or even thousands of genes [4]. In order to take advantage of our developing knowledge of DNA sequences, we need to bridge the gap between those individual differences and their manifestations as phenotypes. In other words, we need to *translate* knowledge at the level of molecules into an understanding of cognition and behaviour [5]. Interactions between the entire genome and non-genomic factors that ultimately result in health and disease, are core components of the new era of 'genomic medicine' [6].

HOW MANY GENES ARE THERE?

Humans have around 19 000 protein-coding genes [6], far fewer than the approximately 120 000 predicted 10 years ago. We possess about the same number of genes as a mouse, and rather fewer than rice plants.

The information content of human DNA is built from long chains of nucleotides. Nucleotides consist of the bases adenine, guanine, thymine and cytosine, combined with a sugar molecule (deoxyribose in DNA) attached to a phosphate group. These bases are usually depicted by the letters A, G, T and C. The nucleotides in a DNA strand are arranged in pairs, and our genome is made up of around 3 billion nucleotide pairs, per haploid set of chromosomes. We normally have 23 pairs of chromosomes, and each member of the pair is identical in females. In males, one of the sex chromosomes is a Y rather than an X, but the total is just the same (46, the full diploid set). To fit such an enormous amount of information into a cell nucleus, the DNA double helix is incredibly tightly coiled. Recent research is focusing on how that coiling occurs, by what rules, and how does the machinery that wants to read the DNA sequence, to make proteins and regulate cellular activity, get access to it? Box 5.1 provides a glossary of basic terms.

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Reading the complete sequence of our DNA for the first time was a mammoth achievement. Let us imagine each nucleotide has the dimensions of an American 1-cent piece, equivalent to a 1 new penny piece in the UK; both have a diameter of about 2 cm. This is of course blowing up the size of each nucleotide enormously from their actual dimensions. If laid down side-by-side, 3 billion pennies would stretch about 35 000 miles, which is more than the circumference of the Earth at the equator (25 000 miles).

SOURCES OF GENOMIC VARIATION

Nucleotides are read in triplets by the cell's genetic machinery, by convention from left to right in any illustration (e.g. AAA, CCC, TTA, ATG). Each triplet codes for an amino acid or a signal of some sort (e.g. start, stop), or it may have no meaning that we currently recognize.

Until relatively recently, we thought that the main reason individuals (of the same gender) differed from one another was due to small differences in the typical nucleotide sequence of their DNA. These changes are known as single-nucleotide polymorphisms (or SNPs for short). On average, our DNA sequence of nucleotides is very similar from one person to another, with only about 0.4% of our genome differing due to these SNPs. Nucleotide substitutions occur on average once in every 800 base (nucleotide) pairs. If the change in our nucleotide sequence (e.g. . . .**T**CTGATTG. . . becoming . . .**A**CTGATTG. . .) occurs in a genetic coding or a regulatory region there may be an impact on gene expression or in the shape of the protein ultimately formed from the gene in question. Alternatively, the substitution may be 'silent' in its consequences.

We call such differences in gene sequence *polymorphisms*, if they are fairly common. Polymorphisms occur by definition in more than 1% of the population. The prevalence of polymorphisms is strongly influenced by the genetic background of the population being studied. This means that their distribution could be very different, for example, in Americans of African and of European origins.

There are other sources of genetic variation too. These include insertions and deletions of relatively small numbers of nucleotides (so-called indels) as well as grosser structural rearrangements within or between chromosomes (the latter can often be detected by microscopy). Where these indels are relatively large (larger than 1000 base pairs) and cause an increase or decrease in the number of copies of a single gene or a series of genes, these are known as copy-number variations (CNVs). Either increasing or decreasing the number of copies of particular genes may alter susceptibility to a variety of disorders. For example, several large-scale studies of autism and schizophrenia [7,8] have shown that particular CNVs are much more common in both conditions than in control populations. Copy-number variations were not recognized as important contributors to risk until very recently [9]. They are more likely to be pathogenic if they comprise deletions rather than insertions of DNA sequences, and may be inherited or occur *de novo*. Their discovery has both challenged existing approaches in psychiatric genetics and opened a new frontier for research, as these CNVs could account for as large a proportion of genetic risk as SNP variation. Determining the implications of a copy number variation for gene function is problematic, because by definition they tend to disrupt large sequences of DNA. The fact that similarly positioned CNVs are found in several major psychiatric conditions seems to indicate the genes have not read the textbooks that so carefully define diagnostic classification [4].

MECHANISMS OF GENOMIC REGULATION

Advances in technology are giving us new insights into the consequences of individual differences in DNA sequences. There is a lot of DNA in every human cell, but rather fewer genes than we expected. Most of the DNA is identical in all humans, but as we have seen, the variability that does exist can have significant consequences for the prediction, prevention, diagnosis and treatment of disease.

Surprisingly, perhaps, we are faced with a number of unexpected and rapidly increasing problems concerning exactly how we define genes, and how they are regulated. We used to think that a gene was a segment of DNA, in the cell nucleus, that coded for a protein, and that the production of that protein was mediated by the action of RNA that read the genetic material and transported the code to protein-building machinery on the ribosome, elsewhere in the cell. In recent years we have found that there are many more classes of

RNA than we ever suspected, and that there is a tremendously complex regulatory machinery.

Genomic regulation is now known to comprise multiple interactions between proteins and RNA molecules, some of which can lead to modifications of RNA itself [10]. Over the next few years, it seems likely there will be an enormous increase in our understanding of the role of small RNA molecules, which are critically involved in this process. Incidentally, they are being discovered at a considerable pace (e.g. 21 microRNA genes had been identified on the X-chromosome by July 2009, but the equivalent figure was 128 just one year later). Unless we understand how individual differences in gene regulation predispose to disorders, we are unlikely to be able to use the information gained from the study of the basic DNA sequence in a human genome to develop models of disease. Risk of psychiatric disorder is not instantiated in a few triplets of DNA nucleotides.

Gene regulation is far more complicated at every level than we ever imagined. Understanding how susceptibility to psychiatric disorder, measured at the level of a small change in a DNA sequence, leads to phenotypic differences at the level of observed behaviour and mental activity will be captured by integrating information from a host of different levels of analysis; from cellular activity to synaptic control, from the efficiency of neural transmission to cognitive processing.

MEASURING GENETIC SUSCEPTIBILITY TO PSYCHIATRIC DISORDERS

Gene—environment interactions

It is self-evident that our genes do not wholly determine our development. Even identical twins, who share 100% of their genetic make-up, are not exactly the same in their personality or propensity to develop psychiatric disorders. But how do our family circumstances, the unpredictable events that happen in our lives, and our genetic-make-up interact? Can we meaningfully predict that some people with a particular genetic predisposition, at the level of a single gene polymorphism, will be vulnerable – but *only* if exposed to risky environments? For instance, is it true that children with a particular genetic variant of the monoamine oxidase A (*MAOA*) gene are much more likely to develop antisocial behaviour in adulthood if subject to maltreatment in childhood than if they did not possess the variant [11]? Should we warn young people who have a polymorphism of the catechol *O*-methyltransferase (*COMT*) gene that they should not smoke cannabis because of a disproportionate increased risk of psychosis [12]?

Do genetic and environmental risk factors combine in ways that lead to a relatively greater probability of outcome than simply the arithmetic sum of the individual risks? The controversial argument from the studies quoted above is that the simple sum of the risks (gene + environmental exposure) on outcome is much *less* than the observed risk. In other words, some interaction must have occurred, between the genetic and the environmental variable that had increased the probability of a deleterious outcome disproportionately, and may imply that these factors had interacted in some way at a biological level.

Interactions between variables are commonly modelled to predict outcomes in epidemiological studies, but many scientists regard gene × environment interactions as tenuous things, which are not necessarily biologically real. Such interactions could instead represent statistical artefacts, and we may not be correct in assuming we can infer biological interactions from statistical analyses of this type [13]. While evidence of non-independence at a physiological level informs how genetic and other risk factors should be modelled in epidemiological studies, the opposite is not true. In other words, we should not infer a biological mechanism from epidemiological evidence of a 'genotype × experience' interaction. Non-linear summation of risks may prompt further investigation as to whether a biological interaction exists, but the observation of an interaction is not conclusive that there is such a mechanism operating. This somewhat sceptical view is reinforced by the observation that many apparently exciting and novel results in psychiatric genetics fail to be replicated, for a variety of reasons including over-optimistic data analysis and publication bias [14]. This issue of non-replication is discussed in the next section.

Genome-wide association studies

In recent years, psychiatrists have been keen to use our newly found knowledge of the sequence of the human genome, and of the genes contained within it (about 1% of the total) to evaluate associations between genetic variation (usually at the level of SNPs) and risk of disease. In principle,

this is simple, and the number of genetic variants that are associated with complex disorders such as attention deficit hyperactivity disorder (ADHD), schizophrenia or bipolar disorder is growing month by month. The basic idea is to test whether a particular genotype is more commonly associated with the disorder than we would expect by chance, given the prevalence of the polymorphism in the general population. There are many pitfalls in the interpretation of such findings, not least the risk of false positive results that do not replicate [14]. Now, independent replication is mandated for studies that aim to be published in leading journals.

It is unusual for the polymorphisms of statistical significance to be found in coding regions, presumably because their impact is on regulatory processes. However, in many cases we simply do not know for sure how identified polymorphisms translate from gene expression to protein synthesis and beyond. Is this a clue that we need to revise our interpretation of the data? Another surprise from the results of psychiatric genome-wide association studies is that each polymorphism of statistical significance accounts for just a tiny proportion of variance in risk. Even in aggregate, the total number of replicated 'risk-associated' polymorphisms accounts for far less of the variance (in, say, the chance of developing schizophrenia) than we anticipated from our knowledge of heritability. The mystery of the 'missing variance' is not peculiar to psychiatric disorders [15]. It has been the subject of substantial recent debate [16]. Clearly, even though we are now able to read the entire DNA sequence, our knowledge about the variety of differences in the genetic code, and their interaction with other factors, remains inadequate at present to explain the observations made at the phenotypic level.

Epigenetic variation

Our chance of developing a psychiatric disorder may also be influenced by changes in the complex regulatory structure that enables genes to be read efficiently, as a consequence of exposure to certain environmental circumstances. Changes in the myriad mechanisms by which genetic activity is regulated but which do not alter the fundamental DNA sequence, are termed 'epigenetic'.

Epigenetic influences on gene expression are almost certainly not heritable. Once acquired, epigenetic marks can nevertheless allegedly change gene expression for life. There are several mechanisms by which this could happen. The most intensively studied of these entails the attachment of methyl groups to specific nucleotides in a regulatory region of the gene, thereby silencing it.

Research in 'behavioural epigenetics' has grown over the past 10 years, centred on McGill University in Montreal, Canada [17]. The excitement of the field is that it offers an explanation for why early adverse experiences might lead to lifelong changes in behaviour. The usual experimental animal is the rat, but some evidence is emerging that epigenetic changes, arising from events in early childhood, could influence human behaviour too [18]. The results of behavioural epigenetic experiments, which characteristically examine the consequence of individual differences in maternal care, are controversial [19].

On the other hand, there is so much interest in the possibility that epigenetic influences could have a role in a wide range of disease susceptibility, from type 2 diabetes to cancer, that a National Institutes of Health sponsored Roadmap Epigenomics Project was launched late in 2010 (http://www.roadmapepigenomics.org/).

THE FUTURE OF PSYCHIATRIC GENETICS: OUR PERSONAL GENOME

A wide range of molecular genetic diagnostic techniques are coming on stream, and these are likely to have increasing importance for the management of individual patients. They are typically based on computer chips, known as microarrays, and such chips can in principle carry a wide range of information. They can currently be used for the study of up to a million polymorphic SNPs at a time (although no doubt this figure will rapidly increase up toward the figure of 24 million bp, which is the amount by which individuals differ on average). They can also capture copy number variations, and epigenetic changes. Chip technology is becoming much cheaper as the economies of scale allow greater production at reducing cost. One application of gene chip technology, which is important for psychiatrists, is discussed by David Mrazek (see Chapter 41, Discovering Psychiatric Pharmacogenomics). He describes how we can tailor treatments to individuals, using the techniques of pharmacogenomics.

Although we may soon be able to obtain, at moderate cost, a copy of our personal genome, the interpretation of the data in that genome is going to be far from simple. When we have '$1000 genomes' (predicted to be before the end of 2012), how will these benefit patients with psychiatric disorders? First, there will undoubtedly be implications for the revision of conventional phenotypic distinctions within and between disorders. We already know that there is shared genetic risk between disorders that were thought to be quite distinct, such as autism and schizophrenia, and that it is theoretically possible to build modelling networks that predict the underlying genetic covariance [20]. Second, we will be able increasingly to understand the origins of mental disorders in terms of dysregulated neural systems. To this end we are beginning to see the development of gene expression atlases for the brain [21]. So far, these techniques have limited applicability to humans, but there is growing evidence that in mice we can use such information to build models linking neural circuitry, regional gene expression, and phenotypic variables such as memory [22].

CONCLUSIONS

We are increasingly able to measure human genetic variation reliably. The cost of providing each one of us with a map of our personal genome is dropping rapidly. When that information is available, it will mark the end of an era where the focus has been on genetic sequencing, and the beginning of a new age in which the functional activity of that genome is front of stage. Inevitably, the world of '*-omics*', exemplified by genomics, transcriptomics, proteomics, epigenomics and so on, will come to impinge on every aspect of medical science. Ultimately, it will influence the assessment and treatment of all conditions discussed in this volume: forewarned is forearmed.

Box 5.1 Glossary of basic terms. Reproduced with permission from NEJM

Allele: One of two or more versions of a genetic sequence at a particular location in the genome.

Base pair (bp): Two nitrogenous bases paired together in double-stranded DNA by weak bonds; specific pairing of these bases (adenine with thymine and guanine with cytosine) facilitates accurate DNA replication; when quantified (e.g. 8 bp), bp refers to the physical length of a sequence of nucleotides.

Complex condition: A condition caused by the interaction of multiple genes and environmental factors. Examples of complex conditions, which are also called multifactorial diseases, are cancer and heart disease.

Copy-number variation: Variation from one person to the next in the number of copies of a particular gene or DNA sequence. The full extent to which copy-number variation contributes to human disease is not yet known.

Deletion mutation: A mutation that involves the loss of genetic material. It can be small, involving a single missing DNA base pair, or large, involving a piece of a chromosome.

Diploid number: Refers to the number of chromosomes in a human somatic cell. Such a cell normally contains 46 chromosomes, comprising two complete haploid sets (see 'Haploid number'), which together make up the 23 homologous chromosome pairs.

DNA: Deoxyribonucleic acid; the molecules inside cells that carry genetic information and pass it from one generation to the next.

Epigenetic change: A change in the regulation of the expression of gene activity without alteration of genetic structure.

Gene: The fundamental physical and functional unit of heredity. A gene is an ordered sequence of nucleotides located in a particular position on a particular chromosome that encodes a specific functional product (i.e. a protein or an RNA molecule).

Gene chip: A solid substrate, usually silicon, onto which a microscopic matrix of nucleotides is attached. Gene chips, which can take a wide variety of forms, are frequently used to measure variations in the amount or sequence of nucleic acids in a sample.

Genome: The entire set of genetic instructions found in a cell. In humans, the genome consists of 23 pairs of chromosomes, found in the nucleus, as well as a small chromosome found in the cells' mitochondria.

Genome-wide association study: An approach used in genetics research to look for associations between many (typically hundreds of thousands) specific genetic variations (most commonly single-nucleotide polymorphisms) and particular diseases.

Genome-wide scan: An assay that measures hundreds of thousands to millions of points of genetic variation across a person's genome simultaneously, either for research or for clinical application.

Genotype: A person's complete collection of genes. The term can also refer to the two alleles inherited for a particular gene.

Haploid number: The number of chromosomes in a gamete; that is, in an ovum or a sperm (in the human this is 23). A somatic cell (anywhere else in the body apart from the germ cells) has twice that many chromosomes (46 — see 'Diploid number').

Human Genome Project: An international project completed in 2003 that mapped and sequenced the entire human genome.

Insertion mutation: A type of mutation involving the addition of genetic material. An insertion mutation can be small, involving a single extra DNA base pair, or large, involving a piece of a chromosome.

Methylation: The attachment of methyl groups to DNA at cytosine bases. Methylation is correlated with reduced transcription of the gene and is thought to be the principal mechanism in X-chromosome inactivation and imprinting.

Microarray: A technology used to study many genes at once. Thousands of gene sequences are placed in known locations on a glass slide. A sample containing DNA or RNA is deposited on the slide, now referred to as a gene chip. The binding of complementary base pairs from the sample and the gene sequences on the chip can be measured with the use of fluorescence to detect the presence and determine the amount of specific sequences in the sample.

Mutation: A change in a DNA sequence. Germ-line mutations occur in the eggs and sperm and can be passed on to offspring, whereas somatic mutations occur in body cells and are not passed on.

Nucleotide: The basic building block of nucleic acids. RNA and DNA are polymers made of long chains of nucleotides. A nucleotide consists of a sugar molecule (either ribose in RNA or deoxyribose in DNA) attached to a phosphate group and a nitrogen-containing base. The bases used in DNA are adenine (A), cytosine (C), guanine (G), and thymine (T). In RNA, the base uracil (U) takes the place of thymine.

Pharmacogenomics: A branch of pharmacology concerned with using DNA sequence variation to inform drug development and testing. An important application of pharmacogenomics is the correlation of individual genetic variations with drug responses.

Phenotype: The observable traits of an individual person, such as height, eye colour and blood type. Some traits are largely determined by genotype, whereas others are largely determined by environmental factors.

Rearrangement: A structural alteration in a chromosome, usually involving breakage and reattachment of a segment of chromosomal material, resulting in an abnormal configuration; examples include inversion and translocation.

Ribosome: A cellular particle made of RNA and protein that serves as the site for protein synthesis in the cell. The ribosome reads the sequence of the mRNA and, using the genetic code, translates the sequence of RNA bases into a sequence of amino acids.

RNA: Ribonucleic acid; a chemical similar to DNA. The several classes of RNA molecules play important roles in protein synthesis and other cell activities.

Single-nucleotide polymorphism (SNP): A single-nucleotide variation in a genetic sequence; a common form of variation in the human genome.

Small (or short) inferring RNA (siRNA): Any short, double-stranded regulatory RNA molecule that binds to and induces the degradation of target RNA molecules.

Systems biology: Research that takes a holistic rather than reductionist approach to understanding organism functions.

Translation: During protein synthesis, the process through which the sequence of bases in a molecule of messenger RNA is read in order to create a sequence of amino acids.

Adapted from Feero WG, Guttmacher AE, Collins FS. Genomic medicine — an updated primer. *New England Journal of Medicine* 2010;362:2001—11.

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Section 1b

General Patterns of Development

6

Clinical Evaluation of Development from Birth to Five Years

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INTRODUCTION

Clinical evaluation of children with behavioural concerns requires considering their physical, emotional, behavioural and social development, alongside relevant environmental influences. Differential diagnosis includes the possibility of organic and developmental disorders with behavioural phenotypes, where developmental markers aid identification. A working knowledge of typical developmental milestones and 'red flag alerts' to possible developmental delay is essential. Required competencies include eliciting parental concerns, taking a relevant developmental history, observing and assessing child development, undertaking relevant physical examinations, and planning investigations and access to local services where further assessment or intervention is required.

CHILD DEVELOPMENT

Child development is a dynamic process shaped by a complex interplay between genetic, biological and environmental factors that operate from the time of conception through to the child's birth and subsequent development [1]. While there is enormous variation in both the pattern and rate of normal development, the sequences of development are similar in most children. Thus, developmental milestones can act as convenient markers for the rate of development.

INDICATORS OF ABNORMAL DEVELOPMENT

Warning signs of possible developmental problems include:

- A rate of development outside the normal range in one or more developmental domains (e.g. echolalia – repetitive imitation of speech – still present by 3 years).
- Absolute failure to develop skills (e.g. absence of multisyllabic and tuneful babble by 10 months).
- Disordered developmental sequence (e.g. advanced reading coexisting with delayed language).
- Motor asymmetry in hand use or walking.
- Developmental regression, i.e. a loss or plateauing of skills.

THE DEVELOPMENTAL EXAMINATION

A developmental examination includes history-taking and observing the child's developmental, interactional and play skills during the assessment process.

History-taking

Prenatal, perinatal and postnatal histories are required, in addition to the family's history and that relating to the family and social environment. Questioning parents or caregivers about broad areas of function elicits concerns and guides

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clinicians on where to focus a more detailed history-taking and examination. The pervasiveness of concerns about the child can be confirmed from information obtained from others such as teachers or health-care staff.

Parents can provide a reliable history when asked open-ended questions and when requested to give examples of behaviour. They are very good at remembering whether or not they had concerns and what these were. Focusing enquiry around transition points, such as starting at a nursery or other significant events, will facilitate parental memory of developmental achievements. Parental *observations* of behaviour (e.g. 'he will fetch his shoes only if they are visible') are generally more accurate than parental *interpretations* of behaviour (e.g. 'he understands everything I say'). Parental views of the causation of any developmental problems need to be elicited allowing parents to be reassured as to any unwarranted concerns that they are responsible in some way – for instance, that maternal depression may have resulted in autism. Parental misconceptions as to other types of causation can also be corrected.

It is valuable to establish from independent enquiry, the reason for any obstetric intervention (e.g. fetal distress), the baby's condition at birth, and any history of neonatal fits. Gestational age should be ascertained for children under 24 months old and consequent adjustments made when assessing age-appropriate behaviours. The child's current health and any history of serious illness may be relevant and should be ascertained.

Observation and interactive assessment

Setting the context: Providing a suitable selection of age-appropriate toys will allow assessment of relevant domains – for example, the child's abilities to copy behaviour (use of rattle); understand cause and effect relationships (using a pop-up toy), define objects by use (hair brush); symbolic understanding (use of doll, tea set); fine eye–motor coordination (use of bricks, cubes), language and play (toys and pictures).

Observing the child: Observations of children's free play and semistructured assessment provide a meaningful and valid ascertainment of children's abilities. A cardinal rule of developmental assessment is to look not only at *what* the child does, but also at *how* s/he does it. Thus, the quality of responses as well as the level of attainments should be monitored.

A range of standardized materials are available to gather detailed normative information for diagnostic or monitoring purposes. Observations need to include the child's attention, emotional state and their initiations and responses. Any change in function and behaviour with increasing task complexity, coping with transition from non-verbal to language-based activity and changes in performance as assessment time lengthens should be noted. Adult help should be kept to a level that allows assessment of the child's own ability to organize their environment and generate ideas.

DEVELOPMENTAL DOMAINS

Children whose sequence of development shows moderate or severe developmental delay, plateauing or regression, require further assessment, including an examination of their vision and hearing. 'Red flag alerts' can be found in Tables 6.1 to 6.5.

Gross motor

Delay in gross motor milestones (Table 6.1) may indicate neurological abnormalities. Although there is a weak correlation between gross motor and general developmental delay, it usually impacts the learning and performance of skills. A physical examination should be combined with assessing the child's sequence of development, style of learning, avoidance behaviours, sensory sensitivities or impairments, as well as a qualitative description of their motor competencies.

Table 6.1 Gross motor milestones.

Developmental milestone	Mean age (months)	Limit age — 'red flag' (months)
Hands open most of the time (not listed)	3	4
Good head control when sitting	4	6
No head lag when pulled to sit		
Sits independently	7–8	10
Walks alone	11–13	18

Visual behaviour, eye—hand coordination and problem-solving

Progressive coordination of maturing vision with head, body and fine motor movements can be observed as the child manipulates a variety of test/play materials. Observation can help tease out the relative contributions of motor and cognitive abilities, emotional factors and experience. Children's achievements here represent the precursor to later non-verbal problem-solving abilities, correlate well with overall intellectual ability and may provide early markers for learning, psychological and psychiatric disorders. Early visual behaviour and eye–hand coordination milestones are presented in Tables 6.2 and 6.3.

Object concepts and relationships

Table 6.4 presents this developmental sequence, which reflects a growing understanding of the nature and visuospatial relatedness of objects, combined with a refined grasp and release ability.

Imitating and copying cube models

This sequence, assessed via children's desire and ability to copy models, combines the processes of encoding, decoding and execution using horizontal, vertical and three-dimensional alignments (Table 6.5).

Drawing

The developmental milestones in drawing ability can be found in Table 6.6. These are evidence that children move through various stages – from simply making marks on paper to the ability to copy a diamond shape by the age of 66–72 months. Children gradually refine their grasp – 50% of children by the age of 3 years and 80% by 4 years have a good tripod grasp of a pencil.

Table 6.2 Early visual behaviour milestones.

Visual behaviour milestone	Limit age (months)	'Red flag' (months)
Visually alert, orient to face	1	Any delay
Visually follows face	2	Any delay
Coordinates eye movements with head turning	3	Any delay

Table 6.3 Eye — hand coordination milestones from 3 months.

Developmental milestone	Mean age (months)
Holds objects briefly when placed in hands without visual regard	3
Visually examines own hand	4
Reaches out with a two-handed scoop	5
Reaches out and grasps objects on table surface with a raking grasp	6
Transfers from hand to hand	6
Explores with index finger	6
Picks up a pellet/raisin between thumb and finger	9
Picks up a string between thumb and finger	10
Can release in a container	10
Has mature grasp	12
Has precise release — without pressing on surface	13
Builds towers of 2 cubes	13
Builds towers to 3 cubes	16
Turns pages of book one page at a time	24

Language and communication

There is a considerable variation between children in milestone attainments as outlined in Table 6.7. Identification of language impairments requires combining parental information with observation and assessment. The quality of parental information is improved if parents keep lists of spoken words or phrases. It is worth noting that as children develop good understanding of daily family routines, their language comprehension can often be overestimated by both parents and professionals.

Play and social behaviour

A number of developmental sequences come together in children's play (Figure 6.1). These include social initiations and responses, verbal and non-verbal communicative skills; imagination and the generation and sustaining of ideas whilst remaining alert to the social environment [2].

Table 6.4 Object concepts and relationships milestones.

Developmental milestone	Mean age (months)
Permanence of object: looks for hidden object	8
'Cast': drops repeatedly-enjoys sound and attention	8
Cause and effect: presses or pushes to activate a toy	9
Means-end relationship: pulls toy placed out of reach with a string	9
Relating two objects together	10
Relating objects: enjoys putting things in and out of container	10
Simple posting games: round shapes	12
Matches simple shapes	16–18
Matches three shapes with good 'scanning'	24

Table 6.5 Cube model copying milestones.

Developmental milestone	Mean age (months)
Builds tower of 6 cubes	22
Horizontal alignment of bricks	24
3-cube bridge	33
4-cube train with a chimney	39
3 steps with 6 cubes	48–54 months
4 steps with 10 cubes	60 months

Some red flag alerts for language, communication and social development

Further assessment would be required when the following 'red flags' for language, communication and social development are observed:

- no multisyllable babble, pointing or other gesture by 12 months;
- no joint attention by 18 months;
- absence of simple pretence play by 24 months;
- no single words by 18 months;
- lack of social interest in other children from 18 months;
- no word combinations (non-echoed) by 30 months;
- cannot follow two-step command (e.g. 'give ball to daddy') by 30 months;
- speech largely unintelligible by 4 years;
- no conversational interchange by 4 years;
- echolalia still present at 3 years.

Development of attention

Attention difficulties may contribute to poor developmental performances and interaction difficulties, or indicate global developmental delay. Difficulties of behaviour, language, learning and coordination often coexist with deficits of attention. A variety of medical conditions in preschoolers, including epilepsy, hypothyroidism, low birthweight, hearing loss and prenatal exposure to teratogens (e.g. fetal alcohol syndrome) may underlie attention deficits.

Attention skills develop sequentially, such that children move from a relatively brief exploration

Table 6.6 Drawing milestones.

Developmental milestone	Mean age (months)
Shape copying	
Makes a mark on paper	15
Scribble	18
Copies lines	24
Copies a circle	36 (90% by 42 months)
Copies a cross	42 (90% by 48 months)
Copies a square	48
Copies a triangle	60
Copies a diamond	66–72
Drawing a person	
Figure with head, other parts, no body	36 (80% by 45 months)
Figure with head, body, limbs	54

Table 6.7 Language and communication milestones.

Developmental milestone	Mean age (months)	Range (months)
Comprehension/receptive language		
Understands 'no'/'bye'	7	6–9
Recognizes own name	8	6–10
Understands familiar names	12	10–15
Definition by use: using objects		by 15
Giving objects on request		by 15
Points to body parts on self/carer	15	12–18
Points to body parts on doll	18	15–21
Identifying objects on naming		by 24
Follows a 2-step command	24	18–27
Functional understanding	30	21–33
Understands prepositions (in/on)	24	18–33
Understands prepositions (under)	30	24–39
Understands action words (e.g. eating, sleeping)		by 36
Understands simple negatives	36	30–42
Understands comparatives	42	36–48
Follows 2 instructions (4 ideas)	42	36–48
Understands complex negatives	48	42–60
Follows 3 instructions (6 ideas)	54	48–66
Expressive language and non-verbal communication		
Jargon	12	10–15
Syllabic and tuneful babble	8	6–9
Pointing to demand	9	
Pointing to share interest	10	9–14
One word	12	10–18
2–6 words	15	12–21
7–20 words	21	18–24
50+ words	24	18–27
2 words joining	24	18–30
200+ words	30	24–36
3–4 words joining	30	25–36
Speech usually understandable	30	30–42
Question words	36	30–42
Pronouns	42	36–48
Uses conjunctions (and, but)	48	36–54
Sentences of 5+ words	48	36–54
Complex explanations and sequences	54	48–66

of toys at the age of 5 months to the flexible, focused and sustained attention of the 3–4-year-old. By 5 years of age children can integrate information from different sources and make and carry out plans with sustained and flexible attention [3].

Cognitive development

Here, toys and developmental task materials are used to assess the child's acquisition of object permanence, their understanding of cause and effect, conceptual and complex thinking, and vocabulary and social understanding. Often

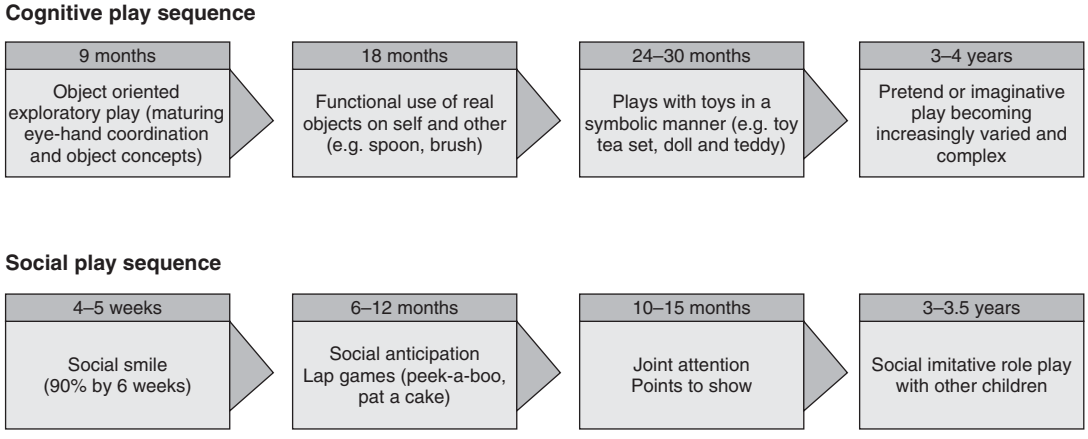
Cognitive play sequence
9 months
Object oriented exploratory play (maturing eye-hand coordination and object concepts)
18 months
Functional use of real objects on self and other (e.g. spoon, brush)
24–30 months
Plays with toys in a symbolic manner (e.g. toy tea set, doll and teddy)
3–4 years
Pretend or imaginative play becoming increasingly varied and complex
Social play sequence
4–5 weeks
Social smile (90% by 6 weeks)
6–12 months
Social anticipation Lap games (peek-a-boo, pat a cake)
10–15 months
Joint attention Points to show
3–3.5 years
Social imitative role play with other children

Figure 6.1 Developmental sequences involved in play and social skills.

Table 6.8 Investigation planning for developmental disorders.

Context	Recommended investigations	Comments
First-line tests		
Children with severe developmental delay or moderate delay that is global or is associated with other significant findings in history and examination	Creatine kinase in boys Thyroid function tests irrespective of neonatal screening Array CGH (if available) Chromosomes for karyotype and Fragile X if array CGH not available MRI scan or CT scan in more severe mental retardation Toxoplasma, rubella assay and CMV urine culture in children under 2 years	Some consensus guidelines also recommend serum lead, urate, U&E, ferritin and biotinidase Microarray-based comparative genomic hybridization (aCGH) is advanced technology that allows detection of even submicroscopic alterations in chromosome balance. These very small changes are often called microdeletions and microduplications. This now incorporates many individual tests, e.g. specific FISH tests and Fragile X. aCGH is receiving increasing support as a routine investigation for generalized learning difficulties/mental retardation (MR). Studies report such chromosome subtelomere rearrangements occurring with a frequency of 7—10% in children with mild-to-moderate MR; approximately 50% of cases are familial. Positive results are eight times more likely than Fragile X in some learning difficulty populations. Plasma calcium and alkaline phosphatase may help with diagnosis and management

(*continued overleaf*)

Table 6.8 *(continued)*

Context	Recommended investigations	Comments
		of DiGeorge syndrome, Williams syndrome, and pseudohypoparathyroidism, and where motor delay is due to vitamin D deficiency
Second-line tests		
The above first-line investigations *PLUS:*		
Associated abnormal head size (micro- *or* macrocephaly), seizures, focal neurological features including severe oromotor impairment and speech abnormality	MRI Where aCGH not available karyotype and specific molecular genetic tests, e.g. looking for 22q deletion in oromotor and speech dysfunction	In some cases MRI studies can show a characteristic signature for metabolic, neurocutaneous and degenerative disorders and can even give enough information to direct subsequent genetic testing
Specific history or examination findings suggestive of neurometabolic disorders	Metabolic investigations: serum amino acids, ammonia, VLCFA, carnitine, homocysteine, disialotransferrin Urine: organic acids, orotate, GAGs, oligosaccharides	Key pointers for metabolic disorders in the clinical history include consanguinity, failure to thrive and episodic neurodevelopmental decompensations (often during minor illnesses). Examination findings may include coarse facial features or hepatosplenomegaly
Specific history or examination findings suggestive of epilepsy or specific behavioural phenotypes, e.g. Angelman syndrome	EEG	In Angelman syndrome, characteristic EEG changes may precede seizures. Diagnosis is confirmed by deletion or uniparental disomy on chromosome 15 Specific EEG changes also may help in rare presentations such as regression in language and differentiation of seizure-like episodes such as in Rett syndrome
Regression with or without associated features	Referral to a paediatric neurologist/ consultation for planning further appropriate investigations	In many countries human immunodeficiency virus (HIV) infections are becoming an important cause of regression with neurological and neuropsychiatric manifestations usually presenting in the first 3 years of life

CMV, cytomegalovirus; EEG, electroencephalogram; FISH, fluorescence in situ hybridization; GAG, glycosaminoglycan; MRI, magnetic resonance imaging; U&E, urea and electrolytes; VLCFA, very long-chain fatty acids.

children presenting with significant developmental delay, global or domain-specific, have long-term cognitive and functional impairments.

CLINICAL DECISION-MAKING AND SEVERITY OF DEVELOPMENTAL DELAY

Developmental delay in any given domain that is equivalent to 50% or less of the expected milestones at a given chronological age is always significant and requires further investigations. Children with less marked ('moderate') delay may or may not have a developmental disorder. The delay is more likely to be significant if global (i.e. across several domains including language and cognition) or where associated with other significant findings or risk factors in the history and examination. If there are any abnormal physical findings such as microcephaly or macrocephaly on neurological examination, then further investigation would be warranted without further delay. The context of a full social, family and medical history and physical examination will help decide whether to watch and wait, promptly investigate, or refer on to other specialists for further assessment and intervention.

PHYSICAL EXAMINATION

The physical examination is generally left to the end of the assessment as any upset to the child would interfere with a subsequent developmental examination. The following key guidelines should be observed:

- *Motor function:* Determine whether there is a motor disorder or if any delay is part of a global learning difficulty. Observe movement patterns and posture during the developmental examination, interaction and play. Formal examination of tone, reflexes and power is largely confirmatory.
- *Symmetry:* Any significant asymmetry of motor skill, tone, reflexes or limb size may indicate hemispheric dysfunction or other pathology.
- *Growth:* Measure head circumference, height and weight and plot on a centile growth chart. Compare the consistency of the parameters and assess the growth rate. Both macrocephaly and microcephaly may be associated with neurodevelopmental disorders. Macrocephaly is more usually familial and it is advisable to measure the parents' head circumference.
- *Sight:* Where examination of the optic discs and fundi is necessary (e.g. for septo-optic dysplasia or raised intracranial pressure) refer to an ophthalmologist or paediatrician. All children with significant developmental disorders should be referred to ophthalmology for this examination.
- *Hearing:* Refer to audiology where there is any delay in language development or significant learning or other developmental disorders. Some sensorineural losses may be progressive; some children will have persistent middle ear problems.
- *Dysmorphism and malformations:* Dysmorphic features and congenital malformations may suggest a particular syndrome or aetiology (e.g. fetal alcohol syndrome).
- *Skin:* Look for pigmented and hypopigmented spots. With significant developmental delay, and especially with epilepsy, a Wood's ultraviolet light examination is required (for ash-leaf skin patches in tuberous sclerosis).

PLANNING MEDICAL INVESTIGATIONS

Part of the responsibility of diagnosing a child with developmental disorder is to consider any potential contributing medical causes and whether any associated or exacerbating condition, such as hypothyroidism in Down syndrome or hearing loss coexisting with autism, is present. Decisions concerning further investigations need to be based on the likelihood of a condition being present, the consequences of a missed diagnosis, the benefits of early diagnosis (e.g. genetic advice) and the consequences for parental planning and coping. There should be a low threshold for getting hearing or vision checks undertaken.

Investigations will depend on the type of developmental disorder and associated findings from the history and examination. The diagnostic yield is highest for global developmental delay with associated history or physical abnormalities on examination, and lowest for isolated developmental problems.

The advent of genetic screening tests with increasing breadth – microarray-based comparative genomic hybridization (array CGH, or aCGH) – has led to a significant increase in positive yield alongside a large number of 'false positives' in the form of benign rearrangements

of DNA picked up by aCGH. Table 6.8 offers an outline of the current evidence-based guidance for investigations.

DEVELOPMENTAL DIAGNOSIS AND MANAGEMENT

Evaluation of a child's developmental abilities requires paying attention to factors that may impinge upon a child's performance, such as anxiety or impulsivity, potentially indicating a need for repeat assessment. Functional difficulties in daily living and the impact of social and biological risk factors are also relevant. Therapy or other intervention may be required even in the absence of developmental delay. Good inter-agency liaison can provide additional information on the child's behaviour in different settings and is essential where children have complex needs. This may reveal concerns regarding a child's protection or care. Early discussions with experienced colleagues and an inter-agency approach are essential for identification or prevention of potential risk to the child.

Finally, a clear management plan is required, incorporating referrals and further investigations, a date for review and identification of a lead/key worker for the family.

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7

Early Social and Emotional Experience Matters: The First Year of Life

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What do infants feel? When, why and with what consequences? This chapter is informed by a functionalist theory of emotion that assumes that emotions evolved as adaptive, survival-promoting processes with intrapersonal and interpersonal regulatory functions [1–3]. For example, Bowlby [4] proposed that fear of the dark, and fear of being alone, are adaptive because there is an obvious link between these events and potential danger. Emotions, in this view, are organizers of personal and interpersonal life, enhancing or restricting development and mental health. The range of emotions a young child feels, and gives expression to, stem from the meaning social interactions impart, and in turn influence the expectations and appraisals one has vis-à-vis the self and others. In other words, the experience of an emotion may be the result of – or the cause of – social interactions. For example, stranger anxiety (which typically appears at 8–9 months of age) may lead an infant to cling to the mother and only feel settled when she holds the baby close saying 'I am here for you'. Through repetition, this kind of interaction will lead the infant to have a sense of trust in the mother, and a hopeful attitude in the face of distress. On the other hand, a different lesson may be learned when infant stranger anxiety is met with an insistence by the caregiver that she must leave, and the baby must manage on his or her own with the stranger. This interaction, if repeated, may accentuate the child's anxiety and contribute to a sense of mistrust in the mother (and others). Other forces that influence infant social and emotional development include the child's biological make-up, in part determined by prenatal experiences as much current research highlights [5], the marital relationship, the wider family network, social economic factors, neighbourhood and broader cultural forces [6,7]. Yet the architect of this multilayered view of contextual influences in child development, Uri Bronfenbrenner, maintained that the family, and in particular the main caregiver (typically the mother) is the filter through which all other influences have their immediate effect.

Early social interactions between caregivers and infants matter greatly because patterns of interaction become established and consolidated over the first year of life into relationship or attachment patterns [8,9] that (i) tend to persist and (ii) have a potentially long-lasting influence on personality and mental health [10] (see also Chapter 15). Six core infant emotional responses are highlighted in this chapter, each calling for sensitive responses from caregivers. Two appear in the neonatal period or soon after – (i) crying and (ii) smiling – and four others appear in consolidated and consistent ways only in the second half of the first year – (iii) sadness, (iv) surprise, (v) anger and (vi) fear. Normal age-related shifts in these emotions are highlighted, notwithstanding individual differences linked to deficits in neurobiological make-up, or social experience. A core take-home message is how all children, whatever their make-up, will thrive to the best of their ability if their social and emotional needs are noticed and responded to in a way that does not overwhelm, or lead to a

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feeling of neglect. Familiarity with the normative sequence of emotional development in the first year of life, outlined in this chapter, may aid the professional and parent alike in knowing how and when to respond to infants' emotional signalling.

CRYING

Newborn babies cry for typically about 30–60 minutes in a 24-hour period. This is about 10–20% of their waking time because newborns (fortunately) sleep for approximately 16 hours, or two-thirds of the day. As with all behaviours, there is a wide range of normal variation, but the 10% of infants who cry for more than 3 hours per day are distressed as much as half of their waking time. This not only causes great concern to caregivers but is also linked to postnatal depression, marital stress and shaken baby syndrome. Fussiness appears to peak at 6 weeks universally [11], but fortunately very cranky newborns typically become much more settled by 3 months of age. And babies whose cries are responded to promptly and efficiently in the first three months, in the context of high marital satisfaction, cry significantly less at 9 months [12].

Infant cries have been reliably identified by scientists, mothers and others as fitting one of three types, indicating (i) hunger; (ii) fatigue or (iii) pain, the latter being a short, sharp, elongated piercing sound followed by apnoea. The hunger cry is one that builds steadily, while the fatigue cry is more of a whimper. Clearly, being in close proximity to an infant helps a caregiver to identify correctly the source of the distress, and responding promptly with sensitivity is the appropriate action.

SMILING OR JOY

The natural course of the smiling response is an instructional illustration of how emotional capacities steadily and gradually appear only partly in response to the quality of care received. In other words, there are well-documented timetables by which certain positive and negative emotions show themselves on infants' faces and in their behaviour. At the same time, the infant's capacity for showing and sharing a wide range of emotions is related to the face the baby sees on the mother or father or other who assumes the responsibility of providing care. Attentive care, including simple verbal descriptions of emotion, in response to infants' emotional expression, is likely to promote children's accuracy in labelling and understanding emotional expressions, and sequences [13,14].

Newborns do not smile, or only appear to smile as when the corners of their mouths upturn slightly in a Mona Lisa way. Such positive expressions are fleeting and appear to indicate sensory comfort, for example following a feed, or the passing of wind, or otherwise becoming used to the good feelings of having some control over being a body in this world. This fleeting positive expression becomes more consistent and definite over the first 6–8 weeks. By 8–10 weeks, there is progression to what is a somewhat more elaborate closed or open mouth smile linked to familiarity with what the infant is looking at, either animate (e.g. mother's face) or inanimate (e.g. a mobile over the baby's crib). For caregivers this is a noticeable advance, and infants of 2 months are frequently said to be smiling. This unfolds into a full social chuckle in the 12–16-week period, completing the initial emergence and organization of the smiling response such that frequent social smiling and laughter are commonly seen only at 4 months. Positive joyful expressions take on an increasingly differentiated range, dependent on the interaction partner. The developmental course of the smiling response appears to be the result of 'hard-wired' neurobiological programming insofar as smiles develop in babies who are born blind. Yet, the smile of the person who has never had sight lacks much of the nuance and complexity seen in sighted people, who have had the benefit (and risk) of the full range of visually perceived social interaction [15].

SURPRISE, ANGER AND SADNESS

Surprise, anger and sadness represent a chain of emotions that result from a functioning memory and set of expectations regarding a hoped for experience or interaction. Surprise, indicated by a vertical oval open mouth and raised eyebrows, is the natural result when things don't appear as they should, or things don't go our way. And when restoration of the hoped for event or interaction does not follow, surprise can quickly turn to protest or anger, with a characteristic furrowed brow and gritting of teeth [16]. And, finally, should this not lead to a successful restoration of the hoped for outcome, resignation and sadness, even

depression, may follow [4]. The point here is that a rather sophisticated cognitive appraisal process underpins these emotional expressions, and it is only in the latter half of the first year that we see definite expressions of these facial expressions of emotion. The caregiver who reads well these emotions on the face of his/her baby will know how valuable it is to speak aloud about the good reasons for feeling these emotions, and the diverse ways of addressing them. Here the point is that research underlines how vital it is to speak to infants, especially from 4 months onwards, in a simple clear way, *describing* what the infant is doing, appears to want, and what one (or others) did or are doing in response. This is the ideal parental response to shared or joint attention [1,13]. In this way, infants will learn the rewards of feeling a range of positive and negative emotions, blended emotions, sequential and mixed emotions, coming to see the natural function and value of emotional experience.

FEAR

Interestingly, the appearance of an organized expression of fear is directly linked to the onset of locomotion around 8–10 months, and the cognitive-motoric achievement of object permanence [17]. With organized knowledge that a valued object can be out of sight, but remains in mind, and can be recovered, infants show stranger anxiety [4], or 8-month anxiety [18]. Fearful protest may bring the caregiver back. Clinically, it is a source of concern when a 1-year-old infant separates *too easily* from a caregiver without protest. Once able to move on their own, infants can easily find themselves in danger, looking over a precipice. Fear is an adaptive response, and one that typically leads to social referencing (looking at the trusted caregiver for cues as to how to behave). The powerful social influence of the trusted caregiver has been demonstrated in classic experiments involving a visual cliff where a crawling infant is placed atop a flat surface that looks (to the infant) as if proceeding would entail falling. It is actually a transparent surface that can support the infant. On their own, infants are typically fearful of the apparently imminent fall, and will not proceed. Yet, when their mother signals to them in a positive way, assuring them it is safe, infants advance, conquering their fear [19]. This effect of trust in the caregiver has been noted repeatedly, particularly when a secure infant–caregiver attachment typifies the pattern of relating. Where fear appears on an infant's face or is indicated by his or her behaviour (e.g. freezing) *in the presence* of the caregiver, evidence suggests that there is a troubling disorganizing element to the child's relationship with the caregiver, one with long-term adverse mental health correlates (see Chapter 15).

The identifiable facial expressions of these emotions – joy, sadness, surprise, anger and fear – were noted by Darwin [2] and then shown to be recognizable around the globe by Ekman and his colleagues [16]. At the same time, the clarity and organization with which infants show these emotions, and later demonstrate verbal labels for them, has been linked to sensitive and responsive care over the first year of life [12,13]. Deficits in labelling emotion faces have been noted during middle childhood for those whose early experience was deficient [20].

CONCLUSION

There is a paradox about early social and emotional development regarding two matters of perhaps equal importance to note: (i) Infants are far more perceptive and competent than was appreciated 50 years ago, calling for respect and sensitivity on the part of caregivers from earliest infancy, if not the moment of conception, forwards, and (ii) yet there is little evidence to support the notion, very popular in 1970, that 'bonding' occurs shortly after birth. The latter notion led to much (over) concern that no mother (or father) should miss out on the '*vital*' opportunity to bond with one's infant in the seconds, minutes and hours after birth – an anxiety-provoking and unhelpful message. Social experience, and attunement between caregivers and infants is vital, but mistakes on the part of caregivers, hopefully not major ones, are inevitable. It is both consistency of care, and reparation following a ruptured, incomplete or confusing interaction [3], that typifies normal social development and optimal mental health outcomes. Professionals and parents alike can benefit from this knowledge that occasional conflict is to be viewed as inevitable and repair/resolution – to be initiated by the caregiver – is seen as essential. A caregiver who invests in reliably repairing ruptures in early infancy, following caregiver

misunderstanding, interference or neglect, is likely to realize the rewards of having a socially competent child in the future, someone capable of establishing and maintaining meaningful and healthy social relationships.

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8

Language Development

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WHAT'S NEW

- Researchers are beginning to explore psycholinguistic processing measures, such as non-word repetition, as clinical markers of language impairment in very young children. Poor performance on these psycholinguistic tasks (measures of verbal short-term memory) is indicative of late language onset ('late talking'). However, the direction of the relationship between memory and language development remains contentious and further work on the earliest phases of language learning will shed light on this issue.
- Usage-based models of language development lead to a more integrated view of what develops when, and holistic assessments of language growth.
- Multidisciplinary work in developmental science (e.g. neural imaging, statistical modelling, behavioural and molecular genetics, computational modelling) will shed new light on the mechanisms and phases of language development.

The ability to communicate using language is one of the most basic human traits. Doing so involves learning to understand and produce an abstract and complex linguistic code that provides the foundation for social interaction, personal relationships, reading and writing, problem-solving, formal learning and personal well-being. By the time children enter school at the age of 4 or 5, most have achieved near adult-like mastery of the sounds and grammar of their native language(s) and are able to communicate with others effectively. This chapter presents some of the main developmental milestones in the language development of preschool children developing in a typical manner, and some of the factors likely to be involved. First, we begin with an overview of the territory.

PROCESSES AND COMPONENTS OF LANGUAGE DEVELOPMENT

The top part of Figure 8.1, adapted from a causal model of developmental disorders [1], illustrates the relationship between language development and some of the factors known to affect it. This simplified model illustrates the interaction between the child's genetic endowment and his or her environment, as would be the case in any area of development. In the case of language development, the human genotype enabled a species-specific endowment for linguistic processing [2], the precise extent of which is still not entirely known. The model also specifies a key role played by the environment, both in the form of human (social) interaction

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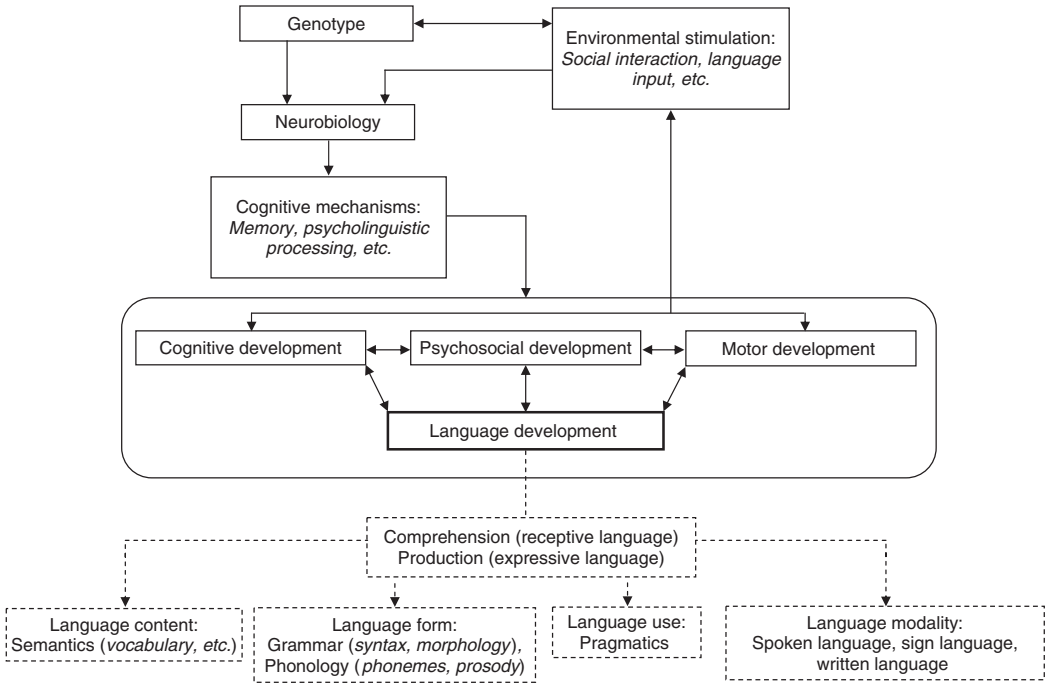

Figure 8.1 Processes, components and modalities of language development.

and language input. The junction of these can be seen in adults' use of a speech register known as child-directed speech – developmentally sensitive speech and language adjustments made by parents and others when talking to children. Both genetic and environmental factors affect the developing brain and its neurobiological mechanisms. These in turn underpin cognitive mechanisms, such as psycholinguistic processing [3,4] and verbal memory [5], which affect how language develops in the child. The child's language ability is also affected by developments in the cognitive, psychosocial and motor domains.

The bottom part of the figure depicts various components and modalities involved in understanding and producing language. Language comprehension (sometimes referred to as *receptive language* in clinical contexts) refers to the process of decoding speech – the chain of events in which sound waves produced by the vocal tract of a human speaker are received, processed and given meaning by a human listener. Language production (sometimes referred to as *expressive language*) refers to the process of encoding – the chain of events by which a message generated by the brain is neurologically relayed to muscles that move the structures of the vocal tract.

In the figure, language is divided into three components: content, form and use [6]. Language content, or semantics, refers to how meaning is conveyed by linguistic elements such as words, idioms and sentences. Language form encompasses grammar and phonology. One aspect of grammar involves the rules governing how words are combined to form phrases and sentences (syntax). For example, the grammar of English requires that the subject precedes the verb (e.g. 'I should go.') in declarative sentences while in questions, the verb precedes the subject (e.g. 'Should I go?'). Another aspect of grammar relates to the structure of words (morphology). For example, in English the past tense form of regular verbs is created by adding the '-ed' inflection to the verb stem (e.g. 'climbed'). Language form also involves the language's sound system (phonology): both its contrastive sound segments (e.g. phonemes such as /p/, /t/ and /k/) and prosodic features such as stress and intonation. Language use refers

to the interpersonal, communicative functions of language during social interaction (pragmatics), such as a speaker's communicative intentions. The model also illustrates various modalities through which language can be communicated, such as its spoken, signed or written forms.

This model is implicitly reflected in current theories of language development, such as usage-based accounts [7,8], in which general learning processes and cognitive mechanisms underpinning language development are mediated by environmental factors. Cognitive mechanisms include such things as pattern recognition and intention reading. Pattern recognition occurs when a child associates new information with something already known, recognizing similarities, such as when the child, knowing that the utterance 'eat apple' describes that event, says 'eat bread' to describe a similar event. Intention reading includes joint attention, such as when the adult and child share a focus on objects, events or interactions, or using pointing and gesturing to indicate objects and events. There is little differentiation between the lexicon and syntax in early language development; rather, the child is learning to express relations in the world, such as 'doggie eat' to describe a dog eating, rather than grammatical relations such as subject + verb. Language development is defined as form-function mappings or construction building.

MILESTONES OF SPEECH AND LANGUAGE DEVELOPMENT

It is beyond the scope of this chapter to present detailed charts outlining children's speech and language development. These can be found on the websites listed at the end of this chapter.

While the sequence of developmental speech and language milestones is broadly similar across children, there is wide variability in terms of when these are reached due to individual differences [9]. Milestones indicate the average age of children when they attain particular features of expressive language such as speech sounds, vocabulary and syntactic constructions, as well as features of receptive language. Both genetic and environmental factors (e.g. the quality or quantity of language input [10]; the presence of developmental delays or disorders), contribute to variations in the attainment of particular milestones. Differences between reported developmental sequences can also be due to differences in definitions or research methods employed by researchers. For example, proposed sequences of speech sound development may be affected by whether children's speech is recorded during spontaneous conversations with adults or during picture-naming tasks when words are elicited singly. It can also be influenced by the methods of data analysis – for example, whether children's speech forms are analysed on their own (phonetic inventory) or in relation to adult forms (phonemic inventory). Further, the national variety (e.g. American, British, Australian or New Zealand English) or dialect (e.g. African American English) of the spoken language will also exert an effect on the acquisition sequence. In what follows, we summarize general patterns of development in typically developing children.

DEVELOPMENTAL PHASES

After 6–7 months of gestation, the fetus responds to sound, including the human voice, and at birth infants prefer to listen to sounds heard before birth, such as the mother's heartbeat and her voice. In the first 6 months of life the infant is highly responsive to adult interaction, intently watching human faces and turning in the direction of sounds. Turn-taking emerges and infants imitate adult tongue protrusion or raspberry blowing and laugh in response to the human voice. By 2 months of age infants can discriminate between phonemes [11] and produce a wide range of speech-like sounds, including those not present in their own language[s].

Between 6 and 12 months the infant's motor and cognitive development advances at a rapid pace. Sitting and crawling afford the opportunity to explore the environment, increasing opportunities for object manipulation and learning about object function, shape and taste. The infant also begins to appreciate the role of others as agents of change, as seen by the use of simple communicative gestures, some of which are accompanied by vocalizations, to indicate and request objects (e.g. pointing) and action (e.g. raising arms to be lifted). At 6–7 months babbling emerges (e.g. 'ba ba ba') and becomes more diversified over the next few months. At around 9 months conventional social gestures emerge (e.g. waving goodbye, shaking head to indicate rejection). At around 11 months infants begin to lose the ability to discriminate among all phonemes, but begin to

discriminate better the speech sounds of their own language environment, fine-tuning their perception to be language-specific [11]. At 10 months the average receptive vocabulary size is about 50 words [12].

By 12–18 months the infant begins to understand the intentions of others, engages in joint attention (sharing attention and focus with an adult) and attends to books for brief periods. Walking now contributes to a greater awareness of self in space. This facilitates the development of comprehension of locative words, such as 'in' and 'on', and simple questions like 'what's that?' and 'where's your teddy?' Parents often report that their child understands most of what is said to them, an impression fostered by the child's use of *comprehension strategies*, which capitalize on context in the absence of understanding exactly what is said [13]. Basic representational play emerges during this phase (e.g. 'drinking' from a toy cup), along with some single words. By now most children can identify some body parts ('where's your nose?') and pictures of family members. By 12 months, children's average receptive vocabulary size is 85 words, increasing to 250 words by 18 months; and by 20 months, some 75% of children are reported by their parents to be combining words [12].

By 24 months representational play includes greater symbolism (e.g. using a block for a car). The toddler shows an understanding of 'what' or 'where' questions (e.g. 'what x doing?', 'where x going?') and some children begin to use the pronouns 'me' and 'you' and grammatical markers such as the '-ing' verb inflection, plurals ('cats') and past tense '-ed'. While the average American toddler has an expressive vocabulary size of around 300 words by 24 months of age, large individual differences exist (7 to 668 words) [12]. Similar findings have been reported in 12 other languages [14]. It is difficult to estimate the size of children's vocabulary beyond the age of 2 years. Psycholinguistic processing mechanisms can be measured at 2 years, with most children being able to imitate one-, two- and three-syllable nonsense words (e.g. 'doe-per-lut') [15].

By 3 years of age the child engages in cooperative play. Phonological awareness and other aspects of metalinguistic ability emerge, as seen in word play (e.g. 'moo', 'goo', 'boo'). Print awareness emerges and the child can point to familiar words in books. The child understands the locative 'under' and begins developing an awareness of causality, accompanied by an increase in asking 'why' questions and producing complex sentences (e.g. 'he's crying 'coz he fell down'), but sometimes with word order errors.

By 4 years, there is greater print awareness and metalinguistic awareness, demonstrated by an appreciation of nursery rhymes. The child begins to ask 'who' and 'where' questions. Play becomes rule-based (e.g. role taking) and the child uses language to organize and talk about their world (e.g. 'you be the mummy'). Sentences contain embedded clauses and are almost adult-like (e.g. 'don't touch that 'coz you'll break it and I haven't finished yet'). The child can now describe basic events, such as their birthday party.

By 5 years the child understands purpose, function and consequence questions (e.g. 'how can we open that jar?', 'what will happen if he loses his keys?'). With knowledge of how objects are oriented in space comes an understanding of more locatives ('in front of', 'behind', 'next to', 'through'). The child can hold a conversation and, with an increase in schema (pattern) knowledge, can describe more abstract events, such as what happens at birthday parties in general or how to make a sandwich.

ATYPICAL LANGUAGE DEVELOPMENT

While language development is very robust for most, some children experience delays or problems in acquiring the sounds, meanings and grammatical structures of their language while others experience difficulty using language socially. Such delays may or may not be transient and are often the first signs of a problem related to hearing, cognition or other developmental domains. They may also be indicative of a primary language disorder (see Chapter 29) or a social communication disorder (see Chapter 23).

Pertinent to this chapter is a group of children with late onset of language, often referred to as *late talkers*. These are 2-year-olds with small expressive vocabularies or no word combinations. Two recent large-scale epidemiological studies have identified several factors associated with late onset of language. In one study [16], male gender, family history of late talking, two or more children in

the family and early neurobiological growth were identified; in another study [17], a family history of speech or language difficulties, non-English-speaking background and low maternal education were identified. While the majority of late talkers will eventually catch up with their age peers, some 30–40% may not. Several smaller scale studies have indicated that poor receptive vocabulary and limited use of gestures may be indicative of more persistent problems but further research is needed. The American Speech-Language-Hearing Association suggests that children should be referred to a speech and language therapist whenever a parent expresses concern (see http://www.asha.org/public/speech/disorders/LateBlooming.htm).

FUTURE DIRECTIONS

Our understanding of language development will be greatly enhanced by advances in, and application of, new technologies and methods to explore neurophysiology, neuroplasticity, human growth models and the synergistic roles of genetic and environmental influences on learning. New techniques in neural imaging, statistical modelling, behavioural and molecular genetics, and computational modelling are being applied to developmental questions. Further innovations and collaborations across physics, pharmacology, biology, psychology, neurology, computer science and engineering will generate new ways of thinking about development, leading to new discoveries.

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INTERNET RESOURCES

Child Development Institute – Language Development in Children: http://www.childdevelopmentinfo.com/development/language_development.shtml

National Institute on Deafness and other Communication Disorders – Speech and Language: http://www.nidcd.nih.gov/health/voice/speechandlanguage.asp#milestones

American Speech-Language-Hearing Association – Typical Speech and Language Development: http://www.asha.org/public/speech/development/

American Academy of Pediatrics Healthy Children: Ages & Stages: http://www.healthychildren.org/English/ages-stages/Pages/default.aspx

9

Development of Social Cognition

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WHAT'S NEW

- Infants as young as 12–18 months have some capacity for mentalizing. Recent experimental studies in which infants spontaneously help, communicate or imitate an adult have revealed that infants can take into account what other people feel, want and know.
- Individual children differ in the rate at which they acquire a theory of mind. Correlational studies indicate that those children who are more skilful at mentalizing are also somewhat more sophisticated and effective in their social lives.
- Environmental factors contribute to individual differences in social-cognitive skill and the most significant correlate is exposure to mentalistic conversation with parents, siblings and friends.

Social cognition in humans is uniquely complex. Unlike other mammals, which respond primarily to each other's outward behavioural signals, we look deeper, into each other's minds, in order to understand one another. For instance, if we witness a stranger breaking into loud song on a crowded street, we might notice that outwardly, she is smiling and acting exuberantly, but our primary reaction would be to interpret her unusual actions by considering what's going on inside her mind. She may want to communicate a message. Perhaps she thinks this is a good way to get a recording contract. Maybe she just feels great. This process of reasoning about what other people feel, want and know, is referred to as mentalizing, mindreading, or using our theory of mind. It is a fundamental skill that helps us to understand and get along with the other people in our social world.

SOCIAL COGNITION IN INFANCY

Until recently, most psychologists thought that the capacity for mentalizing only emerged in the preschool period, because that is when typically developing children begin to pass tests assessing their ability accurately to report what is likely to be in someone else's mind in differing circumstances. Whether or not infants and toddlers also have a theory of mind has been debated for years. Recent experimental procedures, cleverly modelled on everyday situations, now confirm that some basic mentalizing is evident in infants' non-verbal communication, helping and imitation.

For instance, in one experimental procedure, 12- to 18-month-old infants watched an adult write on a piece of paper with a marker. Then the marker dropped off the table, unseen by the adult, who began to search around for it. Already on the floor were some other items, but the infants consistently pointed at or retrieved the marker, rather than the distractor items [1,2]. This showed that 1-year-olds could work out the specific item that the adult *wanted*. In another experiment, 18-month-old infants were invited to interact with two

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adults, who were playing with two different toys. After a while, the toys were put on a shelf and one of the adults left the room. The remaining adult then brought out a new toy, played with it and then put it on the shelf as well. When the first adult returned and pointed toward the three toys on the shelf saying 'Oh look! Give it to me please!' the infants immediately retrieved the new toy – the one that this particular adult had not yet played with. This indicates that the infants interpreted the adult's pointing in terms of what the adult *thought* was new and interesting [3].

Sometimes we witness someone trying and failing to accomplish a simple act. For instance, someone may try to turn on a light switch but her fingers slip off. In this case we automatically mentalize, and see past the external behaviour to the underlying intention: She meant to turn on the switch. Another recent experiment showed that infants as young as 12 months mentalize in the same way [4]. When infants watched an adult try but fail to turn on a switch, they recognized the adults' *intention* and when given the opportunity, they fully turned on the switch. But if the infants watched an adult handle the switch without trying to turn it on, they did not turn it on themselves thus showing that in the first situation, they were genuinely mentalizing, rather than simply doing what might have seemed obvious.

Besides reading the intentions behind each other's actions, we also tend to anticipate each other's intentions and the behaviour they produce. For instance, if you know your friend likes sugar in his coffee, then as he pours himself a cup you are likely to shift your eyes to the sugar bowl, anticipating his mental state as well as his next move. Recent eye-tracking research shows that 25-month-olds anticipate in this way and, furthermore, they can anticipate another person's next move even if that person is actually mistaken. In the eye-tracking study, toddlers watched a video in which an actor repeatedly reached to get his toy out of a box. When the actor wasn't looking, the toy was moved to a different box. Upon the actor's return, the toddlers anticipated his next move and looked to the first box, where the actor still *thought* his toy was located, rather than to where it really was [5]. This experiment shows unmistakable mentalizing because the toddlers focused on the inner experience of the actor, rather than on the actual location of the toy. Another study using precisely the same set-up showed that 6- to 8-year-old children with an autism spectrum disorder failed to do the same thing; they did not look to the box where the actor thought the toy was [6]. This shows that automatic, non-verbal mindreading is disrupted in children with autism, in addition to the more explicit social-cognitive and communicative problems characteristic of the disorder.

SOCIAL COGNITION IN YOUNG CHILDREN

The research described above shows that in typical development, accurate mentalizing is present, in some instances within the second year of life. A direction for future research is to evaluate whether and how these early non-verbal theory of mind skills are linked to the more sophisticated social cognition that develops later in childhood. Although more research is needed, the results of one such study suggest a positive association [7] between infants' imitation of unfulfilled intentions (as in the switch task described above) and their performance on measures of social cognition at 3 years of age.

Soon after children begin to use language, they also begin to talk about what they and others feel, want and think. This facile ability to communicate about what is in our own and others' minds has allowed researchers to expand the range of verbal tests used to assess young children's social-cognitive reasoning. The standard procedure is for children to listen to a description of a social scenario, sometimes accompanied by cartoons or acted out with puppets. Once the scenario is in place, they are then asked questions about what the protagonists feel, want or know, or what they will do next. While there are potentially as many different versions of these tests as there are unique social scenarios, a subset of them has recently been made into a highly reliable developmental scale, illustrated in Table 9.1. The theory of mind scale reveals that 3- to 6-year-old children gradually master different elements of social cognition in a predictable sequence [8,9]. Children with clinical diagnoses that are characterized by delays or deficits in social cognition, such as deafness or autism, pass the tasks in essentially the same order but at a later age. The one exception to scale conformity is that children with autism pass the hidden

Table 9.1 Six tests that make up the theory of mind scale. Research shows that the majority of children acquire these concepts in order from 'Diverse desires' to 'Sarcasm'.

Test	Social-cognitive concept assessed	Approximate proportion of 3- to 6-year-old children who pass
Diverse desires	Different people may like and want different things	95%
Diverse beliefs	Different people can hold different beliefs about the same thing	88%
Knowledge access	People who see something also know about it; if they don't see then they don't know	79%
False belief	People do things based on what they think, even if they are mistaken	49%
Hidden emotion	People can deliberately conceal emotions by facial expression management	27%
Sarcasm	In order to be humorous, people sometimes say the opposite to what they really mean	20%

emotion task before they pass false belief [8,9]. This subtle difference reinforces the conclusion, following from years of research, that the mentalistic concept of false belief is particularly difficult to grasp for people with autism.

INDIVIDUAL DIFFERENCES IN SOCIAL COGNITION: IMPLICATIONS FOR CHILDREN'S SOCIAL LIVES

Alongside the consistent developmental sequence for theory of mind concepts outlined in Table 9.1, there are measurable individual differences amongst children in their rates of social-cognitive development. These individual differences have been linked to some specific consequences for children's everyday social life. Although the effects are typically small to medium-sized, meaning that other factors play a role, children who perform well on theory of mind tests also tend to have relatively sophisticated social skills as well as effective social relationships in their daily lives.

For instance, studies have shown that mentalizing is related to social competence in 4- to 8-year-old children. That is, those children who are good at working out what others feel, want and think, are nominated by their peers as being most likeable and rated by their teachers as being most socially mature [10]. It is important to note that these findings are correlational, so we do not know if skilful mentalizing causes children to be socially adept and popular, or if those qualities put them in the best position to develop their social-cognitive skills.

Other studies indicate that 3- to 8-year-old children who perform well on theory of mind tests are also especially good at keeping secrets, at distinguishing right from wrong in complicated social situations, and at deceiving and lying convincingly [11]. This last point highlights that mentalizing enables children to take part successfully in a wide range of social interactions, including potentially negative ones. Thus acquiring a theory of mind does not necessarily make for a well-adjusted child; indeed, more than one study has revealed that playground bullies, who are often somewhat popular as well as being feared for their manipulative and aggressive interpersonal tactics, possess good or even superior mentalizing skills [12]. Acquiring a theory of mind enables children to understand their social world, but it appears that individual children's temperament and life experiences, among other things, determine how they use that understanding. Predicting how individual children will use their theory of mind – either prosocially

or antisocially – is another important direction for further research [13].

INDIVIDUAL DIFFERENCES IN SOCIAL COGNITION: WHERE DO THEY COME FROM?

Although it is in our nature to look past external behaviour and into each other's minds, the few genetic studies on social-cognitive development carried out to date suggest that nurture is more important in determining individual differences among children. For instance, a major behaviour genetic study comparing 1116 monozygotic and dizygotic 5-year-old twin pairs revealed that the majority of individual variation in the children's mentalizing (based on the kinds of tests listed in Table 9.1) was attributable to environment rather than to genes [14]. This finding contrasted with an earlier, smaller-scale study of 3-year-old twins, which revealed significant genetic influences on mentalizing [15]. More research is necessary to reconcile these findings; however, one possibility is that genes play a role in early social-cognitive development, but by the age of 5, children's theory of mind is shaped primarily by their personal experiences.

One environmental variable that is crucial for theory of mind development is access to language and conversation about people's feelings, desires and thoughts. Mentalizing is consistently correlated with language ability, and there is also a specific link between children's ability to successfully complete tasks like those described in Table 9.1 and their comprehension and production of mentalistic vocabulary. The importance of language to mentalizing is perhaps most evident in the deaf; those deaf children who do not have access to fluent signing partners for daily conversation show social-cognitive delays similar to those observed in children with autism. By contrast, deaf children who have regular access to signed conversation are comparable to typical hearing children in their social-cognitive development [16].

The role of language in social-cognitive development is further demonstrated by the fact that children's theory of mind is consistently associated with their participation in meaningful conversations about emotions, desires and thoughts with parents, siblings and friends [17]. The influence of parents' mentalistic conversation is especially well documented; in a nutshell, the more parents discuss and explain what they and others feel, want and think, the better their children understand those concepts. This principle has been documented in many conversational contexts from everyday disciplinary encounters ('It was really unkind of you to take her dolly; just imagine how it made her feel') to mutual reminiscences ('Remember when the bird stole the baby's fruit bun right out of her hand? She was so surprised and angry!') to book-reading ('Look at that boy's face in the picture; why does he feel so sad?'). It is important to note that the link between children's theory of mind and parents' mentalistic conversation extends to children with autism [18] and deafness [19]. While not yet translated into formal interventions, training studies have shown that exposure to mentalistic conversation boosts social-cognitive skill in typically developing children [20]. Therefore, parents should be encouraged to take the time to discuss feelings and thoughts with their young children; not only will it make for engaging conversation, but it is likely to benefit their children's social-cognitive development.

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10

Social and Emotional Development in Middle Childhood

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WHAT'S NEW

- There is significant continuity in SED from middle childhood to adolescence – psychopathological disorders reflecting problematic SED persist; children who are well adjusted in middle childhood tend to become well adjusted adolescents.
- Recent reviews of early intervention programmes for children at risk of SED problems report significant positive effects in relation to: participants' educational success; social participation; cognitive, social and emotional development; family well-being, and less involvement in the criminal justice system.
- The most effective early intervention programmes for children at risk of SED problems are those that are of long duration and intensive, involving more than 500 sessions.
- Supportive family environments foster social and emotional development and buffer children from the negative long-term effects of bullying.

Social and emotional development (SED) involves the acquisition of skills for expressing emotions, regulating emotions, and managing social relationships within the family, school and peer group [1,2]. Some of the milestones associated with these aspects of development are given in Table 10.1. Middle childhood, the period between 6 and 12 years, occupies a pivotal position between the preschool years and adolescence with respect to SED. During the preschool years rudimentary skills are acquired, while in adolescence, sophisticated skills are refined. However, it is during middle childhood that particularly important developments occur within the emotional and social domains.

THE PRESCHOOL YEARS

During the first year of life there is a gradual increase in non-verbal emotional expression in response to all classes of stimuli including those under the infant's control and those under the control of others. At birth infants can express interest as indicated by sustained attention, and disgust in response to foul tastes and odours. Smiling, reflecting a sense of pleasure, in response to the human

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Table 10.1 Social and emotional development.

Age	Expression of emotions	Regulation of emotions	Managing emotions in relationships
Infancy 0–1 year	Increased non-verbal emotional expression in response to stimuli under own control and control of others	Self-soothing Regulation of attention to allow coordinated action Reliance on 'scaffolding' from caregivers during stress	Increased discrimination of emotions expressed by others Turn taking (peek a boo) Social referencing
Toddlerhood 1–2 years	Increased verbal expression of emotional states Increased expression of emotions involving self-consciousness and self-evaluation such as shame, pride or coyness	Increased awareness of own emotional responses Irritability when parents place limits on expression of need for autonomy	Anticipation of feelings towards others Rudimentary empathy Altruistic behaviour
Preschool 2–5 years	Increased pretending to express emotions in play and teasing	Language (self-talk and communication with others) used for regulating emotions	Increased insight into others emotions Awareness that false expression of emotions can mislead others about one's emotional state
Middle childhood 6–12 years	Increased use of emotional expression to regulate relationships Distinction made between genuine emotional expression with close friends and managed display with others	Increased autonomy from caregivers in regulating emotions Increased efficiency in identifying and using multiple strategies for autonomously regulating emotions and managing stress Regulation of self-conscious emotions, e.g. embarrassment Distancing strategies used to manage emotions if child has little control over situation	Increased understanding of emotional scripts and social roles in these scripts Increased use of social skills to deal with emotions of self and others Awareness of feeling multiple emotions about the same person Use of information about emotions of self and others in multiple contexts as aids to making and maintaining friendships
Adolescence 13+ years	Self-presentation strategies are used for impression management	Increased awareness of emotional cycles (feeling guilty about feeling angry) Increased use of complex strategies to autonomously regulate emotions Self-regulation strategies are increasingly informed by moral principles	Awareness of importance of mutual and reciprocal emotional self-disclosure in making and maintaining friendships

voice appears at 4 weeks. Sadness and anger in response to removing a teething toy are first evident at 4 months. Facial expressions reflecting fear following separation become apparent at 9 months. Infants also show an increasingly sophisticated capacity to discriminate positive and negative emotions expressed by others over the course of their first year of life. During the first year of life infants develop rudimentary self-soothing skills for regulating their emotions such as rocking and feeding. They also develop skills for regulating their attention to allow themselves and their care-takers to coordinate their actions to soothe them in distressing situations. They rely on their care-takers to provide emotional support to help them manage stress. The capacity for turn taking in games such as peek-a-boo develops once children have the appropriate cognitive skills for understanding object constancy. Social referencing also occurs towards the end of the first year when children learn the appropriate emotions to express in particular situations by attending to the emotional expressions of their care-takers.

During their second year infants show increased verbal expression of emotional states, and increased expression of emotions involving self-consciousness and self-evaluation such as shame, pride or coyness. This occurs because their cognitive skills allow them to begin to think about themselves from the perspective of others. During the second year of life toddlers show increased awareness of their own emotional responses. They show irritability – often referred to as the 'terrible twos' – when parents place limits on the expression of their needs for autonomy and exploration. In relationships they can increasingly anticipate feelings they will have towards others in particular situations. They also show rudimentary empathy and altruistic behaviour.

Between the ages of 2 and 5 years children increasingly pretend to express emotions in play and when teasing or being teased by other children. They use language in the form of both internal speech and conversations with others to modulate their affective experience. There is increased insight into the emotions being experienced by others and an increased awareness that we can mislead others about what we are feeling by falsely expressing emotions. More sophisticated empathic and altruistic behaviour also begins to develop within the family and peer group during the preschool years.

MIDDLE CHILDHOOD

During middle childhood (6–12 years), with the transition to primary school and increased participation in peer group activities, children's SED undergoes a profound change. Rather than regularly looking to parents or caregivers to help them manage their feelings and relationships, school-aged children prefer to autonomously regulate their emotional states and depend more on their own resources in dealing with their peers. As they develop through the middle childhood years they show increased efficiency in identifying and using multiple strategies for autonomously regulating their emotions and managing stress. During this process they learn to regulate self-conscious emotions such as embarrassment and to use distancing and distraction strategies to manage intense feelings when they have little control over emotionally challenging situations. There is increased use of emotional expression to regulate closeness and distance within peer relationships. Within this context, children make clear distinctions between genuine emotional expression with close friends and managed emotional displays with others.

During middle childhood children develop an understanding of consensually agreed emotional scripts and their roles in such scripts. There is also an increased use of social skills to deal with their own emotions and those of others. Children become aware that they can feel multiple conflicting emotions about the same person; for example, that they can be angry with someone they like. They use information and memories about their own emotions and those of others in multiple contexts as aids to making and maintaining friendships. As adolescence approaches they develop an increasingly sophisticated understanding of the place of emotional scripts and social roles in making and maintaining friendships.

Cooperative play premised on an empathic understanding of other children's viewpoints becomes fully established in middle childhood. Competitive rivalry (often involving physical or

verbal aggression or joking) is an important part of peer interactions, particularly among boys. This allows youngsters to establish their position of dominance within the peer group hierarchy. Peer friendships in middle childhood are important because they constitute a source of social support and a context within which to learn about the management of networks of relationships. Children who are unable to make and maintain friendships, particularly during middle childhood, are at risk for the development of psychological difficulties.

ADOLESCENCE

During adolescence from 13 to 20 years there is an increased awareness of complex emotional cycles, for example feeling guilty about feeling angry or feeling ashamed for feeling frightened. In adolescence, youngsters increasingly use complex strategies, such as reframing, to autonomously regulate emotions. These self-regulation strategies are increasingly informed by moral principles. However, alongside this concern with morality, self-presentation strategies are increasingly used for impression management. Adolescents gradually become aware of the importance of mutual and reciprocal emotional self-disclosure in making and maintaining friendships.

FACTORS CONTRIBUTING TO SOCIAL EMOTIONAL DEVELOPMENT

Available research indicates that SED in middle childhood is influenced by complex interactions among multiple personal and contextual factors. Personal factors include genetic endowment [3], temperament [4], cognitive abilities [5], self-esteem [6], social cognition [7] and moral development [8]. Contextual factors include attachment [9], parenting style [10], parental adjustment [11], family functioning [12], school environment [13], peer group relationships [14], and the wider social and cultural environment [15]. From a clinical perspective, in any given case, we may expect more successful SED where there are more positive than negative personal and contextual factors. In contrast, where there are more negative than positive personal and contextual factors, problems with SED may occur.

Positive SED

With regard to personal factors, young people are more likely to develop the skills for emotional expression and regulation and for making and maintaining relationships if they have favourable genetic endowments, easy temperaments, adequate cognitive abilities to understand their feelings and the emotional demands of their important relationships, adequate self-esteem, the capacity to understand social situations accurately and a well-developed conscience. With regard to contextual factors, positive SED is more likely where children have developed secure attachments; where their parents have adopted an authoritative parenting style characterized by warmth and a moderate level of control; where their parents have no major adjustment problems; and where the family, school, peer group and wider social environments have been supportive. For example, in a UK study Bowes *et al.* [16] found that children from supportive families showed resilience when bullied in primary school.

Problematic SED

Problematic SED may occur where there are difficulties with genetic endowment, temperament, cognitive abilities, self-esteem, social cognition and moral development. Problematic SED is associated with unfavourable genetic endowments indexed by family histories of psychopathology. A childhood history of difficult temperament is also associated with problematic emotional development. With regard to cognitive abilities, children with intellectual disabilities tend to acquire skills for expressing and regulating emotions and managing relationships at a slower rate than children without such disabilities. Disproportionately more children with intellectual disabilities, than without, show challenging behaviour associated with emotional regulation problems. Children with low self-esteem, who evaluate themselves negatively, have difficulty regulating negative mood states and managing relationships. Children who have problematic social cognition, notably those who have developed a hostile attributional bias where they inaccurately attribute negative intentions to others, have difficulties regulating anger and maintaining positive peer group relationships. Children who have not internalized social rules and norms

and developed a conscience, particularly those with callous unemotional traits, have difficulties empathizing with others and making and maintaining social relationships. The foregoing are some of the ways in which personal vulnerabilities can compromise SED in middle childhood.

Social and emotional development during this period may also be compromised by environmental adversity characterized by difficulties with attachment, parenting style, parental adjustment, family functioning, the school environment, peer group relationships, and the wider social and cultural environment. Problematic SED is more common where children have developed insecure attachments to their parents or caregivers. Non-optimal family environments can also impair SED. Such family environments may be characterized by parenting problems, child abuse or neglect; parental mental health problems or criminality; and family conflict or domestic violence. Where there is a poor match between children's educational needs and educational placement, this can have an adverse effect on their SED. For example, problems with SED may be exacerbated where a child with a specific learning disability, intellectual disability or psychological disorder is placed in a mainstream class without adequate special educational resources. Schools with inadequate policies and procedures for managing bullying and victimization of pupils by peers or teachers can also have a negative effect on SED. Problematic SED may be exacerbated where children are rejected by their peers or where they spend a significant amount of time with antisocial peers. Within the wider social and cultural environment a range of factors can have a detrimental impact on SED. These include a high level of extrafamilial stress and a low level of extrafamilial perceived social support, and also exposure to media (TV, films, computer games) that model and reinforce the inappropriate expression of aggression, anxiety, depression, elation and other emotions.

CONSEQUENCES OF SED PROBLEMS IN MIDDLE CHILDHOOD

Emotional dysregulation is a risk factor for psychopathology [17], and many types of psychopathology and behaviour problems are associated with problematic SED. Anxiety and mood disorders, and internalizing behaviour problems are associated with difficulties regulating fear and sadness. Disruptive behaviour disorders and externalizing behaviour problems are associated with difficulties regulating anger and aggression. Attention deficit hyperactivity disorder (ADHD) is associated with problematic impulse control. All of these types of disorders and behaviour problems are associated with problems making and maintaining relationships, as are other conditions such as autism spectrum disorders and psychoses. There is significant continuity in social emotional development from middle childhood to adolescence. Those who are well adjusted in middle childhood tend to become well adjusted adolescents [2], while problems tend to persist into the teenage years in children who showed social and emotional difficulties in primary school [18].

ADDRESSING SED PROBLEMS

Prevention and treatment programmes have been developed to address SED problems. Successful prevention programmes begin during the preschool years. They involve screening at-risk children on the basis of their status of personal and contextual risk factors, and offering complex interventions such as family support, parent training and child stimulation that target multiple risk factors [19]. With regard to treating children with SED problems, it is best to base interventions in any particular case on a formulation of factors relevant to that specific case and the current evidence base for effective interventions for such difficulties, because problems with SED are typically caused and maintained by the complex interaction of multiple personal and contextual factors [20].

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11

Social-Cognitive Development During Adolescence

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HUMANS ARE EXQUISITELY SOCIAL

Humans are an exquisitely social species. We are constantly reading each other's actions, gestures and faces in terms of underlying mental states and emotions, in an attempt to figure out what other people are thinking and feeling, and what they are about to do next. This is known as theory of mind, or mentalizing. Developmental psychology research on theory of mind has demonstrated that the ability to understand others' mental states develops over the first four or five years of life. While certain aspects of theory of mind are present in infancy [1], it is not until around the age of 4 years that children begin explicitly to understand that someone else can hold a belief that differs from one's own, and which can be false [2]. An understanding of others' mental states plays a critical role in social interaction because it enables us to work out what other people want and what they are about to do next, and to modify our own behaviour accordingly.

THE SOCIAL BRAIN

Over the past 15 years, a large number of independent studies have shown remarkable consistency in identifying the brain regions that are involved in theory of mind, or mentalizing. These studies have employed a wide range of stimuli including stories, sentences, words, cartoons and animations, each designed to elicit the attribution of mental states (see Ref. [3] for review). In each case, the mentalizing task resulted in the activation of a network of regions including the posterior superior temporal sulcus (pSTS), the temporo-parietal junction (TPJ), the temporal poles and the dorsal medial prefrontal cortex (mPFC; see Ref. [4]). The agreement between neuroimaging studies in this area is remarkable and the consistent localization of activity within a network of regions including the pSTS/TPJ and mPFC, as well as the temporal poles, suggests that these regions are key to the process of mentalizing.

Brain lesion studies have consistently demonstrated that the superior temporal lobes [5] and PFC [6] are involved in mentalizing, as damage to these brain areas impairs mentalizing abilities. Interestingly, one study reported a patient with large PFC damage whose mentalizing abilities were intact [7], suggesting that this region is not necessary for mentalizing. However, there are other explanations for this surprising and intriguing finding. It is possible that, due to plasticity, this patient used a different neural strategy in mentalizing tasks. Alternatively, it is possible that damage to this area at different ages has different consequences for mentalizing abilities. The patient described by Bird and colleagues had sustained her PFC lesion at a later age (62 years) than most previously reported patients who show impairments of mentalizing tasks. Perhaps mPFC lesions later in life spare mentalizing abilities, whereas damage that occurs earlier in life is detrimental.

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It is possible that mPFC is necessary for the acquisition of mentalizing but not essential for later implementation of mentalizing. Intriguingly, this is in line with recent data from developmental fMRI studies of mentalizing, which suggest that the mPFC contributes differentially to mentalizing at different ages.

DEVELOPMENT OF MENTALIZING DURING ADOLESCENCE

There is a rich literature on the development of social cognition in infancy and childhood, pointing to step-wise changes in social cognitive abilities during the first five years of life. However, there has been surprisingly little empirical research on social cognitive development beyond childhood. Only recently have studies focused on development of the social brain beyond early childhood, and these support evidence from social psychology that adolescence represents a period of significant social development. Most researchers in the field use the onset of puberty as the starting point for adolescence. The end of adolescence is harder to define and there are significant cultural variations. However, the end of the teenage years represents a working consensus in Western countries. Adolescence is characterized by psychological changes in terms of identity, self-consciousness and relationships with others. Compared with children, adolescents are more sociable, form more complex and hierarchical peer relationships, and are more sensitive to acceptance and rejection by peers [8]. Although the underlying factors of these social changes are most likely to be multifaceted, one possible cause is development of the social brain.

Recently, a number of fMRI studies have investigated the development during adolescence of the functional brain correlates of mentalizing. These studies have used a wide variety of mentalizing tasks involving the spontaneous attribution of mental states to animated shapes, reflecting on one's intentions to carry out certain actions, thinking about the preferences and dispositions of oneself or a fictitious story character, and judging the sincerity or sarcasm of another person's communicative intentions. Despite the variety of mentalizing tasks used, these studies of mental state attribution have consistently shown that mPFC activity during mentalizing tasks *decreases* between adolescence and adulthood (Figure 11.1). Each of these studies compared brain activity in young adolescents and adults while they were performing a task that involved thinking about mental states (see Figure 11.1 for details of studies). In each of these studies, mPFC activity was greater in the adolescent group than in the adult group during the mentalizing task compared to the control task. In addition, there is evidence for differential functional connectivity between mPFC and other parts of the mentalizing network across age [9].

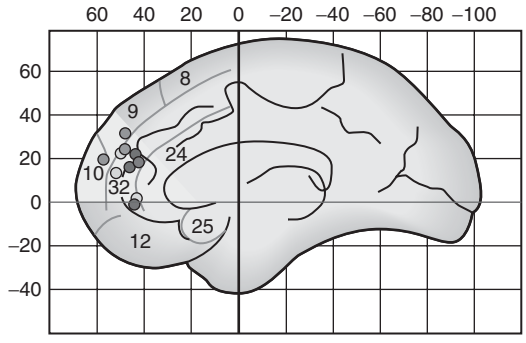

Figure 11.1 A section of the dorsal medial prefrontal cortex (mPFC) that is activated in studies of mentalizing: Montreal Neurological Institute (MNI) 'y' coordinates range from 30 to 60, and 'z' coordinates range from 0 to 40. Dots indicate voxels of decreased activity during six mentalizing tasks between late childhood and adulthood; numbers refer to Brodmann areas (see Blakemore [10] for references). The mentalizing tasks ranged from understanding irony, which requires separating the literal from the intended meaning of a comment, thinking about one's own intentions, thinking about whether character traits describe oneself or another familiar other, watching animations in which characters appear to have intentions and emotions, and thinking about social emotions such as guilt and embarrassment [9]. Adapted from Blakemore [10], with permission.

To summarize, a number of developmental neuroimaging studies of social cognition have been carried out by different labs around the world, and there is striking consistency with respect to the direction of change in mPFC activity. It is not yet understood why mPFC activity decreases between adolescence and adulthood during mentalizing tasks, but two non-mutually exclusive explanations

have been put forward (see Ref. [10] for details). One possibility is that the cognitive strategy for mentalizing changes between adolescence and adulthood. A second possibility is that the functional change with age is due to neuroanatomical changes that occur during this period. Decreases in activity are frequently interpreted as being due to developmental reductions in grey matter volume, presumably related to synaptic pruning. However, there is currently no direct way to test the relationship between number of synapses, synaptic activity and neural activity as measured by fMRI in humans (see Ref. [10] for discussion). If the neural substrates for social cognition change during adolescence, what are the consequences for social cognitive behaviour?

ONLINE MENTALIZING USAGE IS STILL DEVELOPING IN MID-ADOLESCENCE

Most developmental studies of social cognition focus on early childhood, possibly because children perform adequately in even quite complex mentalizing tasks at around age 4 years. This

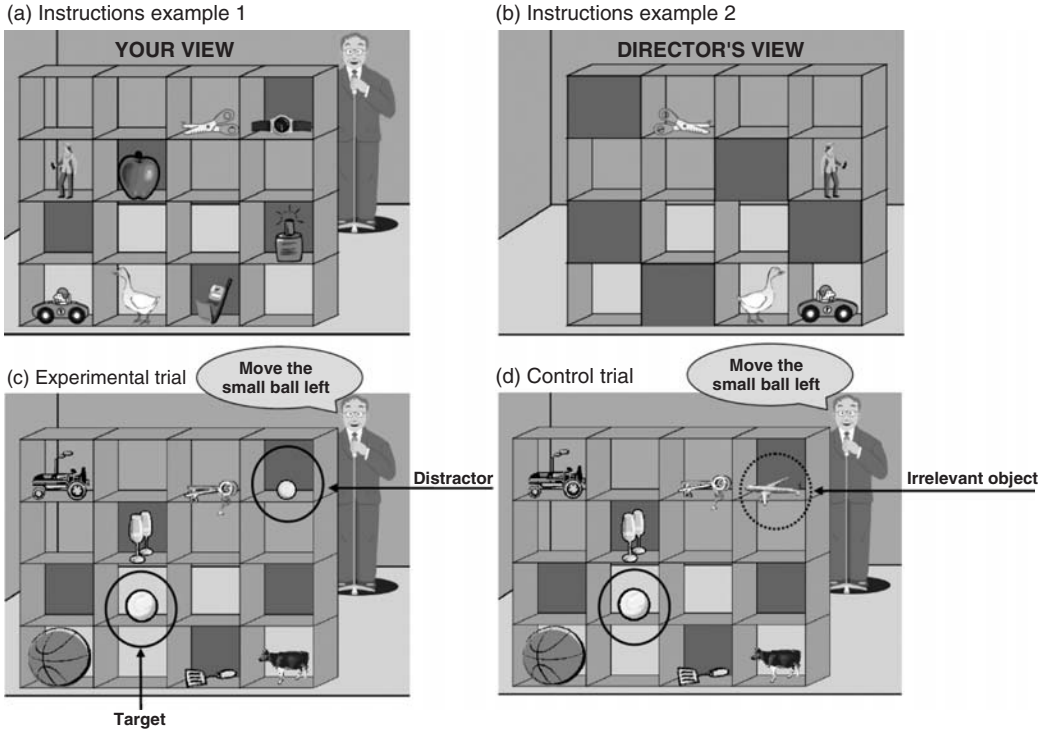

Figure 11.2 (a,b) Images used to explain the Director condition: participants were shown an example of their view (a) and the corresponding director's view (b) for a typical stimulus with four objects in occluded slots that the director cannot see (e.g. the apple). (c,d) Examples of an experimental (c) and a control trial (d) in the Director condition. The participant hears the verbal instruction: 'Move the small ball left' from the director. In the experimental trial (c), if the participant ignored the director's perspective, she would choose to move the distractor ball (golfball), which is the smallest ball in the shelves but which cannot be seen by the director, instead of the larger ball (tennis ball) shared by both the participant's and the instructor's perspective (target). In the control trial (d), an irrelevant object (plane) replaces the distractor item. Adapted from Dumontheil *et al*. [12], with permission.

can be attributed to a lack of suitable paradigms: generally, in order to create a mentalizing task that does not elicit ceiling performance in children aged 5 and older, the linguistic and executive demands of the task must be increased. This renders any age-associated improvement in performance difficult to attribute solely to improved mentalizing ability. However, the protracted structural and functional development in adolescence and early adulthood of the brain regions involved in theory of mind might be expected to affect mental state understanding. In addition, evidence from social psychology studies shows substantial changes in social competence and social behaviour during adolescence, and this is hypothesized to rely on a more sophisticated manner of thinking about and relating to other people including understanding their mental states.

Recently, we adapted a task that requires the online use of theory of mind information when making decisions in a communication game, and that produces large numbers of errors even in adults [11]. In our computerized version of the task, participants view a set of shelves containing objects, which they are instructed to move by a 'director', who can see some but not all of the objects [12] (Figure 11.2). Correct interpretation of critical instructions requires participants to use the director's perspective and only move objects that the director can see (the director condition). We tested participants aged between 7 and 27 years and found that, while performance in the director and a control condition followed the same trajectory (improved accuracy) from mid-childhood until mid-adolescence, the mid-adolescent group made more errors than the adults in the director condition only. These results suggest that the ability to take another person's perspective to direct appropriate behaviour is still undergoing development at this relatively late stage.

Many questions remain to be investigated in this new and rapidly expanding field. The study of neural development during adolescence is likely to have important implications for society in relation to education and the legal treatment of teenagers, as well as a variety of mental illnesses that often have their onset in adolescence.

Box 11.1 Key points

- The social brain is involved in understanding others' minds.
- The social brain develops structurally and functionally in adolescence.
- Activity in medial prefrontal cortex decreases between adolescence and adulthood during social cognition tasks.
- Performance on an online theory of mind usage task improves during adolescence.

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Section 2

Promoting Well-Being

12

Promoting Infant Mental Health

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WHAT IS GOOD INFANT MENTAL HEALTH?

Infant mental health is more than an absence of mental illness, a concept that is difficult in any case to apply to infants. Rather, mental health is a holistic view of the capacities of an infant that include growth, learning and relationships. For an infant it is difficult to imagine an environment in which just one of these might be promoted without also serving the others. A definition that encompasses this perspective is provided by Zero to Three (a multidisciplinary organization that aims to describe and define infant and toddler development):

> *the young child's capacity to experience, regulate and express emotions, form relationships and explore the environment and learn. All of these capacities will be best accomplished within the context of the caregiving environment that includes family, community and cultural expectations for young children. Developing these capacities is synonymous with healthy social and emotional development* [1].

The infant, though physically dependent on its caregivers, has a biological imperative and capacity to react to and interact with other humans [2]. It is now clear that this process shapes not only the learning and emotional capacity of the infant but the very architecture of the brain, with long-term sequelae for infant emotional, social and cognitive growth. Comprehensive guidance on the embedding of infant development in its wider familial, social and cultural context, as well as various avenues to interventions, has been provided [3,4].

WHY IS INFANCY SO IMPORTANT?

It has perhaps seemed paradoxical that the period of 'normal infantile amnesia' for events before the age of about three years could have such a pivotal role in later development. The assumption was that as the child could not remember this period, what happened during that time could not be of any significance. Yet, the evidence suggests that what goes on during this time frame has a powerful predictive value in relation to neuroanatomical and biological processes, as well as social and emotional competence. For instance, Chapter 20 outlines how caregiver abuse and neglect can affect infant brain development.

There is evidence, too, that early exposure to poor parenting leads to abnormal diurnal cortisol patterns, with the normal morning peak and bedtime trough being flattened in children who are exposed to maltreatment. This blunted pattern of cortisol production is seen later in both psychopathy and substance abusers, and may be associated with callous unemotional behavioural traits – a possible mechanism being that under-reactivity of the hypothalamic–pituitary axis (HPA) leads to underarousal at the distress of others. An attachment-based intervention [5] had the effect of normalizing HPA diurnal patterns in children under 2 years, but was less effective with older children suggesting a sensitive period.

The social and emotional development of children may be less obvious than their motor development or their communication skills. Yet, the foundations of social and emotional competence are also laid down in the early months and years

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and have been shown to be the precursors of later social and emotional behaviour, as well as the capacity to sustain attention and learn. There have previously been challenges to critical or sensitive periods in human development because the resilience and flexibility of human learning argued against the sort of tight developmental time frames suggested by animal studies. Early evidence on children raised in institutions however suggested that there were at least some broad limits to tolerance in social and emotional development.

Early research showed that 2-year-old children, adopted from children's homes where caregiver–child relationships were discouraged due to frequent staff changes and high child:caregiver ratios, were at higher risk of later emotional-behaviour problems even after adoption into good homes [6]. More recently, the English and Romanian adoption studies have shown that children who had spent more than their first 6 months in a depriving institution had substantial IQ deficits that were not ameliorated by later good experiences in an adoptive family The children from Romanian institutions were deprived in almost all aspects of their lives, and the duration and extent of deprivation was evident in stunted brain growth, even in the absence of poor nutrition. The authors propose that psychosocial deprivation plays a major role in functional and structural neuroanatomy [7].

So powerful now is the evidence for the impact of the caregiving environment upon infant development, that it has been suggested that since the origins of childhood disruptive behaviour disorders lie in epigenetic processes, intervention as 'near to conception as possible' is the key to effective prevention [8]. The emotional well-being and good social skills that lead to satisfying and sustained peer and family relationships during childhood and adolescence, also lead longer term to patterns of interaction that will later support good relationships with partners, holding down a job and being a parent.

WHAT LEADS TO GOOD INFANT MENTAL HEALTH?

Of course the most fundamental conception of the promotion of infant mental health comes from theories of attachment, as it is within a secure caregiving relationship that infants and then children learn both that they are valued and how to value others. (see Chapters 7 and 15) It is in this secure atmosphere that children can learn and develop to their optimum. Babies cannot modulate their arousal, and depend on an adult to soothe their distress or discomfort when they are overaroused, and to provide stimulation when they are drowsy or underaroused. It is only when the baby is in a state of active attention that it is possible for social interaction and learning to take place. The beginnings of social understanding grow from whether the child is appropriately soothed and stimulated by a carer as required. The child who meets such carefully modulated interaction will learn that other people are trustworthy and available to help. Babies whose needs for emotional modulation are not met will fail to understand their own feelings, fail to read the feelings of others and fail to regulate their behaviour. When arousal is not held in the midrange, that is, it is neither over- nor under-stimulated, learning and thinking are also impaired.

ASSESSING INFANT MENTAL HEALTH

One immediate difficulty emerges in describing or measuring infant mental heath, namely, the lack of clear and predictive measures. The concept of developmental psychopathology has provided some helpful leads to intermediate markers, such as language development, peer relationships and social cognition as well as maternal sensitivity, that are powerful predictors of later good functioning [9]. Even where the marker is something as robust as infant cognitive development, this is difficult to discriminate within the normal range at such an early stage. Due to difficulties of accurate measurement during this period of development, very early cognitive measures do not always reliably predict later cognitive functioning. Less well-defined measures, such as well-being, will depend on the identification of intermediate markers, such as aspects of infant–caregiver interaction.

The gold standard of measurement of infant–caregiver interaction, The Strange Situation [10], is, however, not applicable before about 12 months of age, hence often a proxy or predictor of good attachment, such as measurements of caregiver sensitivity, are substituted. However, screening of development is also a commonly used method of assessment, as it is difficult to imagine

an environment that promotes good development in infancy but would fail to be nurturing in the social and emotional sphere.

Widely used measures of infant mental health include questionnaires for parents, observational methods and rating scales [11]. A recent validation of the Neonatal Perception Inventory has a shown a remarkable continuity of attachment processes, with children of mothers who did not rate their babies as 'better than the average baby' on a number of attributes, showing insecure attachment styles as adults some 30–40 years later [12]. While cognitive development or parent-completed questionnaires of temperament or behaviour may not be perfect indices of infant mental health, for pragmatic reasons they are used in various US states where access to treatment resources may depend on meeting readily accessible criteria [13].

WHAT SUPPORTS GOOD INFANT MENTAL HEALTH?

Universal interventions

In most cases, living in a normal family will provide the baby with exactly the responsive care that is needed to support attachment. The presence of a small number of caring adults who will attend and respond to infant signals reinforces the emotional development of the baby. Some recommendations such as immediate skin-to-skin contact after delivery may have some utility where the relationship is in peril, but these have few clearly demonstrated effects in well-functioning families, although skin-to-skin contact may promote breastfeeding [14]. One intervention that has been shown to be of value is the use of the Brazelton Behavioural Assessment Scale, which was initially designed as a measure of neurological intactness. Since it was used in maternity wards, with mothers present, it became clear that gaining an understanding of the responses of their own baby gave parents a head start in early relationships via understanding what their baby was like and how they could best soothe and support him or her. This was further enhanced by the use of diaries, which prompted parents to observe their babies closely and led to the development of the Touch Points programme [15].

There are a number of universal or population-based interventions that might be described as public health interventions and that can have considerable impact. The use of baby slings and backward-facing buggies which give the infant close proximity and the chance to interact with the carer, are simple low-cost interventions that require little or no professional input. Other relatively simple interventions have been shown to have a significant impact on parent–child attachment. Baby massage has been suggested to have some effect on mother–infant interaction, sleep relaxation and stress hormones, though no direct effect on infant attachment [16].

Families with indicated additional needs

A sound meta-analysis of controlled intervention trials [17] has identified common aspects of effective interventions that increase parental sensitivity or attachment for families where additional support is needed. The common features are that programmes focus specifically on attachment rather than less specific support. It is evident that fewer than five sessions may not be sufficient to produce change, while participating in more than 16 sessions has diminishing value. The ideal time to intervene appears to be between 6 and 12 months of age, a finding that is coherent with finding of the sensitivity of the infant to severe deprivation or abuse at that age. The review cites no direct impact on sensitivity for antenatal intervention, though engaging very needy mothers-to-be antenatally and for a further 2 years has a substantial payoff, perhaps through the development of a working therapeutic alliance [18]. While most research was conducted with mothers, similar findings were shown where intervention was with fathers, though including fathers and mothers together diminished the effect size for mothers. Video feedback proved to be a powerful tool and increased effect sizes for intervention.

A number of programmes meet these criteria, notably Video Interaction Guidance, Circle of Security Attachment, and Biobehavioral Catch-Up. Mellow Babies, Watch, Wait and Wonder and Parent-infant Psychotherapy also showed the potential to improve attachment relationships, cognitive development and emotion regulation in infants [16,19,20]. Inevitably, these intensive programmes require trained practitioners, with training being both expensive and time consuming.

Fortunately there is no need for specialist psychological services for most families. A normally loving family with a few caring adults involved in the regular care of a baby will almost

always provide the interaction that a baby needs. The resilience of the human infant allows a tolerance of a range of parenting. Where parental factors such as postnatal depression, or factors in the baby such as prematurity, or social factors such as poverty or teenage pregnancy, reduce the availability of attuned responsiveness, then specialist intervention is needed. However, we now have very good indicators of how best to intervene and the evidence to promote the earliest possible intervention.

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13

Promoting Children's Well-Being

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WHAT'S NEW

- School-based prevention programmes provide an opportunity to widely promote and maintain the psychological well-being of children.
- The effects of these programmes are, however, variable with those focusing upon anxiety showing more promise than those focusing upon depression.
- Targeted programmes produce greater immediate treatment effects although the potential benefits of universal prevention programmes in maintaining healthy status and reducing the prevalence of new disorders have seldom been assessed.
- Variability within studies indicates the need for methodologically robust research to identify important mediators and moderators.
- An analysis of costs/benefits and delivery models is required to determine whether school-based prevention programmes should be widely available and are sustainable in schools.

Community surveys highlight that psychological problems in children and young people are common and can significantly impair everyday functioning [1,2]. If left untreated, problems persist and increase the likelihood of psychological problems in adulthood [3,4]. Improving the mental health of children is an important public health objective and although effective treatments are available the majority of children, particularly those with emotional disorders, remain unidentified and untreated [5]. Focusing upon the treatment of established disorders will therefore have a limited impact upon the psychological health of children.

An alternative approach is that of prevention, which aims to reduce the prevalence of psychological problems and disorders while optimizing psychological well-being. This can be achieved by the widespread provision of programmes designed to reduce or mitigate the effects of known mental health risk factors while enhancing protective factors at the individual, family and community level. Prevention programmes, therefore, help children to become more resilient and better able to cope with stress and adversity thereby maintaining their healthy status.

PREVENTION

Prevention programmes are typically conceptualized as universal, selective or indicated, with each having a different focus and aim [6] (Table 13.1). Universal programmes are provided to all

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Table 13.1 Universal, selective and indicated prevention.

Prevention	Provision	Advantages	Disadvantages
Universal prevention, e.g. anxiety prevention programmes for 9/10-year-old children	Universal — provided to all regardless of risk status	Far-reaching coverage Opportunity for primary prevention, i.e. reduce prevalence of new disorders Screening not required Avoids need for labelling, which could be stigmatizing Low cost/high volume	Limited resources used to provide interventions to many who are, and will remain, 'healthy' Intervention effects are typically small Face validity, relevance and engagement can be difficulties
Selective prevention, e.g. anxiety prevention programmes for children where parents are separating	Targeted — upon those at increased risk of developing problems through exposure to known risk factors	Resources focused upon 'at-risk' groups Opportunity for primary prevention	Potentially stigmatizing Difficulties in accurately identifying 'at-risk' groups within the community
Indicated prevention, e.g. children with significant anxiety symptoms but not meeting full diagnostic criteria	Targeted — upon those displaying mild/moderate problems	Efficient use of limited resources Provide early interventions for those with emergent problems Demonstrate larger treatment effects	May require screening, which can be costly and practically complicated Potentially stigmatizing and unacceptable to some of the identified group

members of the target population irrespective of risk status, such as children of a certain age. Selective programmes target children at increased risk of developing problems through exposure to known risk factors – for instance, children of parents with a mental illness. Universal and selective programmes are primarily concerned with promoting well-being and in reducing the occurrence of new problems. Indicated programmes are early interventions provided on a targeted basis to those already displaying mild or moderate problems to prevent them from worsening – for example, children with symptoms of anxiety or depression.

Each approach has strengths and limitations. Universal programmes offer the greatest potential to optimize the well-being of the wider population. They provide opportunities for prevention (e.g. maximizing potential), protection (e.g. developing competencies) and intervention (e.g. minimizing impairment). Far-reaching and accessible, they minimize any potential negative stigma arising from more targeted approaches. However, their general focus may not be of sufficient depth or dosage to benefit those with more established disorders. Similarly, from an economic perspective, many of those who receive universal interventions are already healthy and do not, and will not, require any intervention to maintain this status or maximize their potential.

Selective and indicated approaches are more targeted, focusing limited resources upon those with potentially greater needs. The effects are often large since initial levels of symptoms and the subsequent change are greater. However,

they require accurate identification of the target group – a particular difficulty where children have emotional problems or disorders.

School-based prevention

In terms of delivering prevention programmes schools provide convenient and familiar locations that are attended by the majority of young people. The integration of emotional health programmes within the school setting and curriculum offers the potential to discuss openly mental health issues and to promote psychological concepts and ideas as 'skills for life'. This open and more visible approach serves to normalize common psychological problems such as anxiety and depression, and can help to develop a supportive peer group culture where worries and problems can be more openly acknowledged and discussed.

In terms of effectiveness, systematic reviews of school-based emotional health prevention programmes have found evidence to suggest that universal and targeted/indicated approaches can have positive effects upon emotional well-being although the results are variable [7,8]. This chapter focuses on interventions aimed at two of the most common emotional disorders, namely anxiety and depression. Issues and challenges involved in delivering effective prevention programmes are presented and discussed.

DEPRESSION PREVENTION PROGRAMMES

A Cochrane review of depression prevention programmes identified 18 psychological interventions of which 10 were universal and eight were targeted [9]. The methodological quality of the studies was poor resulting in half being excluded from the analyses. Interventions were found to be effective in comparison to no intervention, with significant reductions in immediate post-intervention depression scores. The authors suggest further investigations are warranted alongside methodologically robust investigations. A more recent review identified 42 trials assessing 28 different programmes [10]. Of these trials, 26 were universal, 10 indicated and six were selective. The majority (76%) of programmes were based upon cognitive behavioural therapy (CBT) and involved eight or more sessions (88%). Two-thirds were led by graduate students, mental health practitioners or teachers. Indicated programmes were most effective in reducing symptoms of depression, with prevention programmes led by teachers tending to be the least effective. The authors noted variability in the effectiveness of programmes based upon the same theoretical model suggesting that factors other than the programme content or mode of delivery (universal vs targeted) per se may be important mediators of outcome.

The effectiveness of universal prevention programmes was investigated in a review of 12 studies [11]. The results were variable. Five showed significant immediate post-intervention improvements on at least one measure of depression, but none showed any significant effects at follow-up (i.e. 12 months or longer). The authors concluded that the widespread use of universal depression programmes in schools would be premature. They advocate that further research should be undertaken. The authors highlighted a number of issues, many of which were addressed in a recent randomized trial involving 5634 adolescents in which a CBT-based intervention, 'beyondblue', was compared with no intervention [12]. 'Beyondblue' was delivered by trained teachers and provided interventions at individual, school and community levels. Individually adolescents learned skills to improve problem-solving, social skills, resilient thinking and coping strategies. Within the school the intervention aimed to build a supportive environment by improving social interactions and facilitating access to support and professional services. Finally, community forums were provided in order to facilitate a greater understanding of emotional problems and how to seek help. This multi-level intervention, delivered over 3 years, failed to find any significant differences in depressive symptoms when compared with the 'no intervention' group. This study provides a timely reminder of the difficulties of implementing psychological interventions in everyday settings.

Finally, Horowitz and Garber (2006) suggest that evaluation of depression prevention research has focused upon demonstrating evidence of treatment effects (i.e. reducing levels of depressive symptoms) rather than on preventive effects, such as a reduction in the emergence of new cases. Their meta-analysis of 30 studies found that selective and indicated programmes were more effective than universal programmes. Only four studies provided any evidence suggesting a preventive effect [13].

ANXIETY PREVENTION PROGRAMMES

The results of school-based anxiety prevention programmes are more consistent and encouraging. A recent review [14] identified 27 trials assessing 20 different programmes; 16 universal, eight indicated and three selective trials were evaluated. The majority (78%) were based upon CBT interventions that were mainly led by mental health practitioners (44%) or teachers (26%). Only four studies included children under the age of 9 years. Seventy-eight percent of interventions reported significant post-intervention reductions in symptoms of anxiety, with universal and targeted programmes being judged equally effective. There was considerable variability in effectiveness within individual programmes. Unlike depression prevention interventions, teacher-led anxiety prevention interventions were equally as effective as those led by mental health professionals. The authors suggest encouraging the widespread implementation of school-based anxiety prevention programmes, alongside rigorous evaluation of their longer-term outcomes.

'FRIENDS for life' is one of the better evaluated anxiety prevention programmes. The 10-session programme is based upon CBT and has versions for children (aged 7–11), youths (12–16) and more recently for young children aged 4–6 years (Fun FRIENDS). The programme is very engaging and involves a mix of large and small group work, role plays, games, activities and quizzes, and teaches children skills in three main areas. Cognitively, children are helped to become aware of their anxiety-increasing cognitions and to replace them with more helpful and balanced cognitions. Emotionally, they are helped to understand the anxiety response and their unique physiological reaction to stressful situations. This helps children to detect early signs of anxiety so that they can intervene to manage and reduce these unpleasant feelings. The final component addresses the behavioural domain and teaches children problem-solving skills and the use of graded exposure to systematically face and overcome their worries. FRIENDS can be led by trained teachers or mental health practitioners such as school nurses or psychology graduates.

In addition to the child sessions, parents are invited to two to four psycho-educational sessions. These help parents to understand anxiety and to develop strategies to cope with their own anxiety. They are also taught problem-solving and the principles of contingency management and reinforcement in which the child's courageous and coping behaviour is rewarded rather than their anxious talk and problem avoidance.

Randomized controlled trials have demonstrated significant post-'FRIENDS' reductions in anxiety, which have been maintained up to 3 years after the intervention [15,16]. Similarly, the issue of effectiveness when delivered within everyday settings has been assessed in a series of small studies, with gains being present for up to 12 months [17,18].

FUTURE DEVELOPMENTS AND CHALLENGES

Whilst school-based prevention programmes offer the potential to improve the psychological well-being of children, further research is required before their widespread implementation can be advocated. Firstly, from a methodological viewpoint, sample sizes are often small, medium-term follow-ups are lacking, and few have included comparisons with other active interventions. Most studies have focused upon adolescents and few have been designed for, or included, children under the age of 9 years. In terms of programme content, those based upon CBT, particularly for anxiety, show most promise although there are considerable differences between programmes in length, core components and delivery. Variations in effectiveness within similar programmes suggest the importance of mediating factors relating to programme leaders (e.g. professional experience, training, rapport and confidence), students (e.g. age, gender, engagement) and schools (e.g. class size, emotional health awareness and available support).

Secondly, it is unclear whether preventive interventions that are universal are more effective than targeted interventions. Universal programmes have the potential to maximize psychological well-being. Typically such programmes aim to reduce symptoms, in addition to maintaining psychological health. However, the focus of evaluation has been on whether they reduce symptomatology, rather than on detailing whether they maintain emotional well-being and protect children from subsequent emotional health problems. The longer term primary preventive

benefits of universal approaches need to be assessed alongside an economic evaluation to determine the costs/benefits of such approaches.

Thirdly, integrating emotional health prevention programmes within schools poses many practical issues that will impact upon their sustainability. In terms of effectiveness, most studies are efficacy trials, and the applicability and benefits of these programmes when delivered under less controlled everyday conditions are unclear. Flexible programmes that can be delivered within the teaching timetable and within the length of the school semester are required. In terms of programme leaders, trained school staff appear to be effective in delivering anxiety prevention programmes but less so for depression programmes. Whether depression programmes require a higher level of expertise and understanding, or whether students are less willing to acknowledge or engage with teachers about more personal issues relating to low mood requires further investigation. Attention needs to be paid to the skills and training of the programme leaders and the ongoing supervision that will be required to maximize effectiveness and maintain intervention fidelity.

Finally, whilst there are a number of anxiety and depression prevention programmes available, very few have been subject to robust multiple evaluations by different research groups. Defining the characteristics of effective and sustainable prevention programmes is important. Undoubtedly these will include a number of factors such as multi-level, developmentally sensitive, engaging interventions that rest upon evidence-based conceptual frameworks. These need to be delivered flexibly by appropriately trained and supervised leaders and be consistent with the ideologies and priorities of the schools involved [19].

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14

Fostering Resilience in Adolescents

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WHAT'S NEW

- There has been a shift from looking at variables correlated with resilient outcomes and processes such as attachment and support networks to understanding individuals dealing with adversity within resilient systems.
- There is an increasing understanding of the importance and role of cultural processes.
- Future directions will integrate multidisciplinary knowledge gained from genetics, neuropsychology and cultural psychology about resilience in the developing individual.
- New forms of intervention are moving from promoting resilient individuals to mobilizing basic protective systems at the individual, family, community and organizational level.
- 'Prevention science' seeks to prevent or limit damage at all levels of resilience-supporting systems.

INTRODUCTION

There is increasing social concern about 'out of control' adolescents who exhibit challenging behaviour. Many who come to the attention of the mental health system have experienced multiple adversities in their lives, such as poverty, chaotic parenting, residential care, foster care or school expulsion. They live in systems that lack supportive capacity and are resource impoverished. Significantly, these adolescents are also difficult to engage in therapeutic interventions, particularly because of a fear of stigmatization. There is a developmental challenge inherent in this situation – the health-seeking part of the psyche is outward looking, and strives towards self-efficacy and autonomy; yet the prospect of engaging in therapy may reinforce unconscious fears of being 'mad' or a 'psycho'.

A traditional clinical approach to this group of young people would be that of healing psychopathology. Fostering resilience offers an alternative lens and involves a fundamental shift from a deficits perspective, focused on individualized negative functioning and vulnerability, to a dynamic systems, strengths-based, participative orientation. This chapter outlines new theoretical resilience frameworks and links them to a psychotherapeutically informed participatory action research project with 'hard to reach' adolescents in an exploration of how resilience can be fostered in practice and community settings.

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WHAT DO WE MEAN BY RESILIENCE?

Resilience can be defined as 'patterns of positive adaptation during or following significant adversity or risk' (Masten *et al*. [1], p. 118). The definition requires two judgements – first, that the exposure to risk or adversity was serious enough to pose a serious threat to healthy development; second, that the individual subsequently meets age-related developmental tasks. Within this framework, there has been a focus characterizing resilient outcomes as individual variables (e.g. effective problem-solving skills, emotional self-regulation, positive parenting) or processes (e.g. attachment patterns, family and community interactions). One of the most significant shifts in resilience science over the past decade has been a new emphasis on resilient *systems*. Here, resilience is seen to be mediated by risks, protective factors and resources at multiple levels ranging from the molecular neurobiological to the social, cultural and political [2]. An illustration is provided by analyses of resilience to trauma and stress. One of the most consistent research findings has been that close emotional relationships and supportive informal and formal social networks foster resilience [1,3,4]. Neurobiological research indicates that emotional support of this kind can reduce uncertainty and stress by impacting upon the hypothalamic–pituitary–adrenal (HPA axis) and the sympathetic nervous system, which regulate cortisol levels. This in turn enhances coping via an impact on brain regulatory systems that control arousal and thus behavioural and cognitive responses to stress [5] (see Chapter 20). Masten and colleagues suggest that systems operate at multiple levels and include powerful motivational systems such as the mastery system as well as relational systems at the level of family, community and society, and that the greatest threat to children's resilience may be adversities that damage or undermine these basic human protective systems [1].

Agency in resilience

Of course, cultures influence definitions of resilience, and this is explored in Ungar's multi-country resilience project [6,7]. In many cultures, psychological explanatory models of distress are uncommon, and causality is more likely to be attributed to external social or structural factors. For Ungar, therefore, resilience involves both the capacity of individuals to navigate their way to obtaining health-sustaining resources (including opportunities to experience feelings of well-being) and the capacity of the individual's family, community and culture to provide the needed resources and experiences in a way that is culturally meaningful [6].

Mobilizing social networks to foster coping and resilience

A further useful analysis of how social relationships foster resilience is provided in the Social Convey model [8]. Social convoys are the multiple relationships in the lives of children and young people that facilitate the exchange of affective support, self-affirmation and direct aid. Importantly, the model extends the concept of attachment relationships to other close relationships and acknowledges that relationships between adults and children are characterized by mutuality of support and social exchange. That is, in adult–child relationships, children and youth have the capacity both to give and to receive nurturance and support, and furthermore this is a powerful motivational force in relationship formation and maintenance. The model is strengths-based as it posits an engaged young person who is active in reciprocal support relationships. This may be particularly relevant for adolescents engaged in the developmental task of negotiating a balance between autonomy and relatedness in relationships [9].

IMPLICATIONS FOR POLICY AND PRACTICE

The models outlined above indicate the importance of supporting the agentive, help-seeking, mastery-oriented capacities of young people as they negotiate the support needed from those around them. Several challenges face practitioners if these models are to be successfully applied. Practitioners need to foster adolescents' capacities for: mobilizing adaptive support systems; negotiating access to resources for healthy growth and development; and participating in social convoys characterized by reciprocal supportive relationships. They need also to foster resilience in situations when adolescent support systems may be damaged, unsupportive or unavailable, as is often the case with the families of 'hard to reach' adolescents. Finally, they need to shift

Box 14.1 Promoting resilience — a participatory action research (PAR) project.

PAR project phases	Description	Fostering resilience
Phase 1: Defining participation; question-posing; data gathering; analyses	Twelve weeks of creative arts workshops facilitates: • Exploration of participants' experiences with police, youth justice and social integration • Identification of priority issues • Opportunities to socialize • Exploration of important themes • Meeting key policy-makers to discuss priority issues • Choice of social action project	Sharing of daily hassles and difficulties, and co-constructed art results in: • Enhanced emotional regulation • Communication skills • Information-processing • Behaviour respectful of group members and facilitators
Phase 2: Planning a social action	• Control of budget line • Plan and implement social action project • Choose method of action and dissemination (produce a DVD)	• Develop trust in their ownership of key project decisions • Enhanced motivation • Enhanced reflective capacity • Responsible participation
Phase 3: Implementing social action	Skills development culminating in girls' DVD production '*Girls Out Loud*', including: • Undertaking social action — visiting other relevant projects to learn about consultation with 'key' players, e.g. politicians and community leaders • Make videos of project visits • Interview other young people in social projects • Use interviewing skills in visit to political representatives in Parliament Buildings, Northern Ireland Assembly • Identification of further research questions and themes • Design and art skills — videoing and photography; drawing; storylines; animated puppet shows; video editing	The production of the storylines and DVD mobilized: • Individual and collective mastery • Confidence and self-esteem • Persistence in the face of doubt and difficulty • More powerful voice (making oneself heard) • Effective communication

PAR project phases	Description	Fostering resilience
Phase 4: Analysis, reflection and dissemination of project findings	Present DVD and discuss issues about the justice and care systems with: • Ombudsman for Children • Garda Juvenile Diversion service • local police • local school • local communities Subsequently local youth organization starts participation initiative, consulting with parents. Our project participants: • train staff • develop consultation work with parents	Changes in: • Perspective-taking and inter-subjectivity • Ability to adopt the perspective of the 'other' in their communications

away from an emphasis on internal psychological processes and clinician/patient-defined interventions, and become one part of the adolescent's total resilience system. In practice, this means initiating processes that facilitate young people in defining their own needs, priorities and best interests and in mobilizing their social networks and communities to support them as they address those needs.

MOBILIZING RESILIENCE: AN ILLUSTRATIVE EXAMPLE

Here I outline a participatory action research (PAR) social integration project with nine girls (aged 12–18 years), half of whom had received formal cautions from the police, and who had also been referred to an intensive support service for young people in crisis. A number were in foster or residential care, and some had actually been ejected from the latter. The remaining participants were community peers without formal contact with the police and who were not in crisis. Facilitator participants included a psychotherapist, a creative artist, and two peer researchers from the same community. The author, a psychologist, was principal investigator.

Summary of the project

The intervention project unfolded in three phases over 24 weeks (Box 14.1). It took the form of weekly creative arts workshops in which experiences of police, youth justice and social integration were explored. We anticipated that the girls would undertake a leading role in planning and decision-making: they would choose the art medium to work in; they would choose, design and implement a social action project that reflected their primary issues of concern; they would control an allocated budget for their project. Throughout, the girls interacted with the outside world in ways determined by them – for instance, through visits to other social projects and meeting politicians, community leaders and other key actors to question them about matters of concern. Their final social project – a DVD outlining their issues with the justice and care systems, and social integration – was presented to Ireland's Ombudsman for Children, senior members of the

Garda Juvenile Diversion service, local police, schools and community organizations.

Implementing the project

Some of the developmental stages of this project are outlined below.

- *Getting the girls to engage with the project* was a key challenge – their experiences of feeling powerless and unheard within the social care and justice systems were evident in an early comment by one young participant:

  > *If you feel you're not being heard, there's no point, you feel there is no point in yourself making progress*

  Such experiences fed into a manifest reluctance to join in early workshop sessions. It was clear that the girls felt they had nothing to contribute and found it hard to imagine a project directed by them without adults structuring and controlling it. We tried engaging them in various ways. For instance, we included a drumming session to provide structure and focus while participants learned about the project and each other. It was difficult to get them to drum making audible sounds. It often seemed as if the young people would disengage with the project and that it would be impossible for the group to find direction. This was evidenced in late arrivals, much leaving and returning to the room and a lot of mobile phone texting.
- *The need to step back* and leave the control and ownership of the project in the girls' hands quickly became apparent, and gradually they became more engaged. For instance, they swiftly assumed control of choosing and ordering the end of session food; they developed their own rules for group meetings, including that members needed to arrive ahead of time so they could chat together before the group meeting started; they chose the sessional art medium they would use. First, however, they 'interviewed' the group's creative artist, seeking information about his work, examining examples of it, and asking questions. The group agreed that he was 'sound' and they could work with him.
- *Emerging group level properties* gradually become evident as the girls chose art activities, exchanged helpful ideas and began to work alongside each other.
- *Individual and collective mastery developed*. Initially, participants were reluctant to use the arts materials – one was so inhibited at the start that the creative artist held her hand to scaffold her early drawing attempts. A 'transformational' came when the creative artist used the clay characters that participants had made in a previous session to make an animated computer film. This created great enthusiasm and excitement. From then on, participants' assurance in their contribution to the group grew.
- *A sense of ownership* of the group had developed by session 6: members arrived on time, they reminded each other to turn off mobile phones and they more obviously helped each other, particularly if someone had missed a session. There was a sense of focus and flow. The group assumed significant responsibility for managing their session. One asked 'how many weeks have we left?' indicating how they valued the space. One requested that no new people should be allowed to join as 'this is the group now'. Over the next 6 weeks, their creative work and discussion about their lives opened up. They developed a puppet show and took charge of developing storylines. They began photographing their work.
- *Participants underwent remarkable changes* as they gained in confidence, self-esteem and optimism. Emotional control and regulation became apparent – in particular, inhibition of disruptive behaviour. They showed the capacity to plan and think ahead. Relationships between group members and with the facilitators came to be characterized by reciprocity and commitment to the achieving group aims.
- *Mobilizing supportive resilience systems* flowed from the girls' progress and development. As they moved from being angry and antagonistic towards authority figures, such as the 'pigs' (police), they were able to engage constructively in discussions with them. Their new found ability to tolerate multiple perspectives (theirs and those of the police) and increased maturity meant that when they showed their DVD to local police, their schools and local community projects, they were mobilizing supportive relationships that would enhance resilience within their immediate microsystems.

CONCLUSION

Specific examples of the girls' enhanced resilient capacities are outlined in Box 14.1. In summary, our experience gained through this project has shown us that fostering resilience with 'hard to reach' adolescents means giving them a good or *positive* experience of power, control, ownership of decision-making and resource management in a way that stimulates their feelings of mastery (their mastery system). They gain a sense that they are able to impact on their world in ways that are chosen by them and meaningful to them, and that they can actively mobilize others to support them (Box 14.2). These processes, so important in developing resilience when in difficult circumstances, may inadvertently be undermined by many of our more traditional clinical practices. As researchers and practitioners, our moment of enlightenment in this project came when the young people told the Irish Ombudsman for Children 'This was *our* project' and their sense of ownership was publicly celebrated and claimed.

Box 14.2 Implications for clinical practice

Fostering resilient systems adds multiple layers of complexity to clinical practice. It also positions the clinician and mental health organization as one element of the adolescent's resilience system. Within that, what is the role of clinicians in fostering resilience in the system?

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Section 3

Attachment and Separation

15

Attachment Theory: Research and Clinical Implications

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WHAT IS ATTACHMENT?

Broadly speaking, attachment refers to the tendency, particularly but not exclusively, of infants and young children to rely on a parent figure for comfort and support when frightened, stressed or ill. It is thought to be a form of biobehavioural adaptation, shaped by the forces of natural selection to maximize survival and eventual reproduction, and the key features of attachment are similar across many mammalian species, particularly the higher primates. The field of attachment owes much to John Bowlby [1], who articulated an evolutionary account of attachment, and Mary Ainsworth [2], who pioneered its study in naturalistic contexts.

It is important to distinguish between attachment *behaviour* and an attachment *bond*. It is generally recognized that one cannot classify a behaviour as an attachment behaviour from its outward appearance alone. Instead, attachment behaviours are defined as such by recognizing their function. They are thus any organized, systematic behaviour that is triggered by the appearance of a potential threat or stressor and that predictably serves to achieve proximity to a selected caregiver [3]. This means that all manner of behaviours can serve the general purpose of achieving comfort and security for children, and some may be quite idiosyncratic to a particular child.

Attachment behaviours generally are divided into three classes: (i) signalling or distal communication (e.g. calling, crying); (ii) proximity seeking (e.g. crawling, walking, reaching) and (iii) contact maintenance (e.g. clinging). Monitoring the whereabouts and availability of an attachment figure may also be described as attachment behaviour. Attachment is characterized by *heterotypic continuity*, meaning that while it evidences continuity over time in its basic functional organization, the specific child behaviours used to achieve comfort or security change radically in complexity and sophistication as children mature [4].

Thus, attachment behaviour performs a kind of homeostatic function (Figure 15.1), and to operate efficiently it needs to be guided by information about the environment, for example the nature and location of the threat, the caregiver's whereabouts, and contextual information as to the potential efficiency of various forms of action, etc. Developing this idea, Bowlby proposed that during repeated experiences within an attachment relationship, children develop internal working models of attachment that guide their thinking, feeling and behaviour in attachment situations, and this shapes the way they approach close relationships – and see themselves within them – in the future.

An attachment bond [5] refers to the longer-term, stable tendency to seek out a selected parent figure in times of stress. The processes that lead to the establishment of a long-term attachment bond are quite different to those that trigger attachment behaviour. Crucially, certain forms of disturbance

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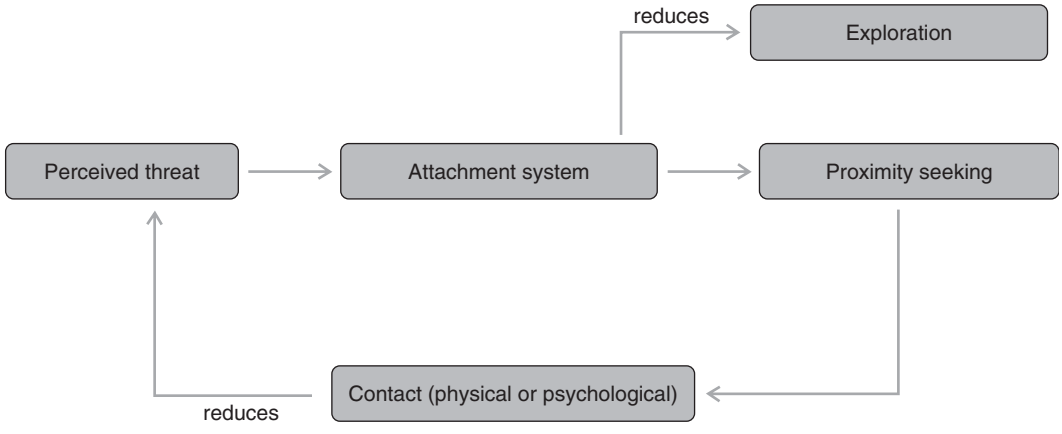

Figure 15.1 Schematic diagram of the homeostatic function of attachment.

in attachment probably result from disruptions in the formation of attachment bonds (e.g. disinhibited attachment disorder), while others result from experiences or influences that alter how attachment behaviour is organized and triggered (e.g. disorganized attachment).

ATTACHMENT VARIATIONS AND THEIR MEASUREMENT

Normative attachment patterns

Mary Ainsworth's Strange Situation Procedure is the most commonly used tool for studying attachment behaviour [2]. Involving an encounter with a stranger and 2–3-minute separations from a parent in an unfamiliar setting, it is used with infants aged approximately 11–18 months. The reliable and valid Ainsworth coding system quantifies four dimensions of attachment behaviour, each rated on a seven-point scale. It also enables raters to make categorical judgements about the style or classification of attachment behaviour displayed by individual children (Table 15.1). These divide into two broad classifications – 'secure' (Type B) and three types of 'insecure' attachment: Type A (Avoidant), Type C (Resistant) and Type D (Disorganized). These classifications have become a major focus of research, with findings indicating that the majority of infants in low-risk circumstances (approximately 65%) are described as 'secure'; approximately 15% as Avoidant; 10% as Resistant and 15% as Disorganized. It is this latter category that has attracted the most attention clinically, as it appears most closely related to more severe forms of adverse parental care, and to raised risk of psychopathology [6]. The prevalence of the different insecure subtypes varies considerably across cultures [7]. A host of similar measures have been developed for assessing attachment in older children [8].

CAUSES OF VARIATION IN ATTACHMENT

Ainsworth originally proposed that the extent to which the parent was sensitive and responsive to the child's attachment signals was the critical determinant of attachment security (see Table 15.2 for exemplars of sensitivity) [2]. Since then, this hypothesis has been supported by an impressive database of longitudinal studies [9]. Furthermore, randomized controlled trials of clinical interventions designed to improve sensitive parenting have been shown to increase the likelihood of secure attachment, suggesting the association is a causal one [10]. However, many authors have noted that the effect sizes in correlational studies or clinical trials are not large and that other factors – either different aspects of parenting or different types of causal influence altogether – probably play a role. While sensitive care may be thought of as the most important proximal determinant of attachment security, a host of more distal or contextual factors also appear to be consistently associated with security and insecurity, including parental depression, social support, marital quality

Table 15.1 Attachment behaviour rating scales and classifications for the Strange Situation Procedure [2].

Interactive behaviour scales	
Proximity seeking	The intensity and persistence to make contact on reunion with the caregiver. An infant scoring high on this scale makes a purposeful approach to the caregiver and takes initiative to make contact
Contact maintenance	The persistence in maintaining contact with the caregiver once it is achieved. A high score on this scale is given when an infant displays resistance to being put down (e.g. clinging), persistent efforts to remain close to the caregiver, or any sign the infant is not ready to terminate contact (e.g. a sinking embrace to mother)
Resistance	The intensity and duration of angry behaviour and resistance of contact directed towards the caregiver. Examples include pushing away, batting away, arching back, squirming to get down
Avoidance	The intensity, duration and promptness of attempts to avoid contact/interaction with the caregiver. Examples of avoidance are averting gaze, moving away and ignoring the caregiver
Disorganized/ disoriented	Anomalous behaviour, e.g.: sequential or simultaneous displays of contradictory behaviour; undirected, misdirected or incomplete behaviours; stereotypies, freezing, disorientation; fearful responses in presence of the caregiver
Infant attachment classification profiles	
Secure	Secure infants use the caregiver as a secure base for exploration and as a source of comfort when needed. The infant is visibly aware of the caregiver's absence at separation. At reunion, the infant greets the caregiver with an approach, smile, gesture or vocalization and seeks contact with the caregiver if distressed. Contact is comforting and infant is able to return to play
Insecure-Avoidant	An avoidant infant appears to be more interested in the environment than the caregiver throughout the procedure. During separation from the caregiver the infant is typically not upset. Upon reunion, the infant will ignore or actively avoid contact
Insecure-Ambivalent	An ambivalent infant prefers to maintain contact with the caregiver than to explore the environment, or exploration is limited. During separation the infant will be distressed. At reunion the infant displays angry behaviours towards the caregiver and/or is inconsolable. Contact with the mother is not effective in regulating the infant's state or supporting a return to play
Insecure-Disorganized	The infant's behaviour lacks an organized, coherent strategy in relation to the caregiver. See above for behavioural descriptions

Table 15.2 Scales of parenting sensitivity and frightened/frightening behaviour.

Maternal sensitivity scales [2]	
Sensitivity vs insensitivity	The degree to which the infant cues are perceived, responded to promptly and appropriately. A sensitive parent is able to empathize with the infant's experience, promoting accurate interpretation of the infant's cues, resulting in appropriate and flexible responding
Cooperation vs interference	The degree to which participation in the infant's ongoing experience is gentle, co-determined and supportive, rather than harsh, overwhelming, directive or controlling
Availability vs neglecting	The degree to which the parent is physically and psychologically available to his/her infant. An available parent is perceptually alert and responsive to the infant even in the face of distraction or his/her own thoughts and feelings
Acceptance vs rejection	The degree to which the parent is able to integrate the joys and stresses of being a parent, as expressed in her/his behaviour toward the child. An accepting parent will not direct, nor attribute negative feelings towards their child or become irritable, enabling her/him to maintain a positive and accepting stance towards the infant
Anomalous parenting behaviours [14,15]	
Frightened or frightening parental behaviours	Behaviours towards the infant that are: threatening, dissociative (e.g. 'spacing out'), frightened, timid/deferential, spousal/romantic or disorganized
Disrupted affective communication	Behaviours that when displayed, particularly during times of stress, can result in unmodulated fear/arousal in the infant (e.g. contradictory signalling to the infant about the caregiver's availability, failure to respond to infant cues; displays that the caregiver is frightened by the infant; hostile/intrusive behaviours; dissociative and withdrawing behaviour)

and poverty [11]. Importantly, evidence indicates that genetic factors play a quite limited role in the development of attachment in infants and preschoolers (see, e.g., Ref. [12]).

Disorganized attachment has a quite different set of determinants than the other insecure categories. Critically, maltreatment has been consistently linked with Disorganized attachment [13]. Furthermore, in populations where rates of maltreatment are likely to be low, observed sensitivity (or insensitivity) appears not to be a critical factor [13]. Instead, a quite different set of parenting features has been implicated, representing behaviour that has been described as frightened/frightening or extremely insensitive [14] (Table 15.2). Aside from the great clinical significance of these findings, they also provide support for an intriguing theory about the causes of disorganized attachment behaviour originally proposed by Main and Hesse [15]. They argued that the incoherent behaviour seen in disorganization occurs when the parent is the source of both comfort and threat. This is thought to create an irreconcilable approach–avoidance conflict, which causes disruptions in attachment behaviour (see Table 15.1), as two incompatible tendencies compete for control of behaviour. The data on frightening parenting and maltreatment bear this idea out well, although the precise mechanisms just described have never been directly verified.

Attachment disorders

Disorders of attachment have been found when children have experienced either the complete absence of a consistent carer, severe maltreatment or major disruption in the continuity of care – as in children raised in institutional or foster care. A significant number of children raised in such circumstances show quite pervasive patterns of disturbed social relatedness, collectively referred to as reactive attachment disorders (RAD). The current diagnostic systems – the *Diagnostic and Statistical Manual of Mental Disorders IV, Fourth Edition – Text Revision* (DSM-IV-TR) and the *ICD-10 International Classification of Mental and Behavioural Disorders in Children and Adolescents* (ICD-10) – define two types of RAD. The first, known as the inhibited/withdrawn type, is marked by extreme withdrawal, a lack of a clearly preferred attachment figure, a pervasive tendency not to seek comfort from others when distressed, and a lack of social responsiveness or reciprocity. The second, known as the disinhibited type, by contrast is marked by indiscriminate attachment behaviour and friendliness and a lack of wariness of strangers (e.g. wandering off with strangers). There are a number of tools for assessing attachment disorders and related behaviours, including standardized questionnaires, interviews and observation schemes [16].

It is critical to note that RADs are quite distinct from the normative patterns of attachment described in the previous section, both in terms of the behaviours that define them and the circumstances that appear to give rise to them. Existing evidence suggests that normative attachment patterns represent variations in the organization of attachment related to the style or quality of parenting among children who have formed one or more selective attachment bonds. In contrast, disinhibited attachment disorder most likely represents the failure of the establishment of a selective attachment bond in the first place [6]. The picture is a little less clear for the inhibited-type attachment disorder, but it may occur when a child is able to form some selective attachment bonds that are subsequently severely disrupted.

CONSEQUENCES OF VARIATIONS IN ATTACHMENT

Early attachment relationships are thought to exert a significant and important influence on current and later relationships, well-being and psychological health. Longitudinal research suggests that securely attached children may have developmental advantages over their insecure counterparts in areas such as emotional regulation and understanding, social cognition, social competence and emotional/behavioural problems. While not all findings have been consistently replicated, recent meta-analyses have found robust associations with peer relationships and externalizing problems [17,18]. With respect to the latter, the evidence indicates that Disorganized children are the most at-risk amongst the insecure subtypes [6,17].

The jury is still out on whether the effects of early attachment on later development represent the direct influence of early experience, or whether continuities over time in other intermediary processes (within the individual and in their environment) are responsible. Considerable evidence suggests that some of the effects of insecurity fall into this latter category. For example, continuity in the quality of care is associated with longitudinal links between attachment and outcome [19]. On the other hand, some findings do suggest that early experience can have specific and lasting effects, particularly severe early deprivation and its effect on disinhibited attachment disorder and accompanying symptoms [6].

INTERVENTIONS

Two broad types of attachment interventions are apparent in the literature. The most widely used are those in the realm of prevention. Here, the aim is to improve rates of secure attachment with a view to promoting resilience and reducing the risk for later emotional or behavioural problems. The second type of intervention focuses on children where attachment problems are primary, or of great clinical relevance – for instance, children who have experienced maltreatment and may be in foster care, or late-placed national or international adoptees.

Preventive interventions

An example of a highly successful preventive intervention is that conducted by van den Boom [20], in which 100 highly irritable neonates were allocated at random to a treatment or control group. Home visits to mothers and infants in the treatment group focused on maternal interaction skills, encouraging

mothers to respond appropriately to their infant's cues, encouraging soothing in response to infant distress, and increasing playful interactions. Large positive effects on maternal sensitivity, and on infant attachment security, were found and these were maintained at a 3.5-year follow-up. A meta-analysis of interventions aiming to increase maternal sensitivity and promote secure attachment in low-risk community samples or at-risk clinical samples (e.g. maternal postnatal depression) has been undertaken [10]. The authors found that the most effective interventions were those that were:

- less than 16 sessions in length;
- were behaviourally oriented and focused on sensitivity (rather than support, or parental representations);
- targeted clinical populations;
- began after age 6 months.

Critically, intervention effects on attachment were strongest when the intervention successfully improved sensitivity, and when the treated population had a large percentage of insecure infants, suggesting, perhaps not surprisingly, that appropriate targeting is important in achieving successful outcomes. Sensitivity-based interventions have also been shown to be effective in reducing rates of Disorganization [21]. While the majority of interventions concern infants and toddlers, some very promising treatments are available for preschoolers and older children (e.g. the Circle of Security programme; see Ref. [22]).

Interventions with fostered and adopted children

A number of effective treatment packages have been devised that are specially tailored for foster care and adoption (see also Chapter 17). For instance, the Attachment and Biobehavioural Catch-Up programme is a 10-session multi-component intervention addressing mutual processes between parent and child that may interfere, directly or indirectly, with the child's self-regulatory capacities and attachment. These include: parental interaction skills; parental attributions; and how the parental childhood history may contribute to current parenting attitudes and behaviour. This approach has been found to improve attachment behaviour and normalize stress patterns as indicated by the hormone cortisol [23].

CONCLUSIONS

The study of attachment has highlighted the potential importance of understanding the early relational roots of both adjustment and maladjustment. The field has also focused attention on how one observes and measures attachment and the sometimes subtle interactional processes that take place within parent–child relationships. Critically, this has led to the development of a range of quite effective treatment techniques designed to enhance the security of attachment relationships in early life. The long-term effectiveness of such treatments for reducing risk for psychopathology and promoting resilience remains to be fully established, and is an important area for future clinical research.

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16

Children Bereaved by Parent or Sibling Death

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Bereaved children grieve in similar ways to bereaved adults, reporting shock and disbelief, followed by sadness, anger, a longing for the dead person to return, and difficulties with concentration, sleeping and eating [1].

CHILDREN'S UNDERSTANDING OF DEATH

There is a developmental progression in children's understanding that death is permanent, irreversible and implies the complete cessation of bodily function. Preschoolers believe, and act as if, a dead person can return. Until the age of 7 years, children believe their thoughts and feelings can cause or reverse death. Around the age of 11 years, when the concept of death is fully understood, children cease worrying about the deceased being cold or lonely, although they imagine an afterlife where the dead remain sensate and enjoy favourite activities. This can comfort some children who believe their dead parent 'watches over' or cares for them. Adolescents can be troubled by the unfairness of death and existential questions as to the meaning of life [2]. A full understanding of the concept of death is acquired faster in cognitively and verbally able children or those who have known a person die previously.

HOW CHILDREN EXPRESS GRIEF

The ability of children to distract themselves from grief through normative activities such as play or social activities can lead their carers to wonder whether they truly grieve. This uncertainly is compounded by young children's inability to verbalize their feelings. Children's curiosity about the death will take new forms as they mature.

Early childhood

Young children will search actively for the deceased. Their play and fantasies reflect their particular concerns and preoccupations. Their sense of loss, their carer's grief, and changed daily routines can provoke bewilderment, developmental regression and unprovoked expressions of anger or aggression.

Middle childhood

Appropriate sadness exists alongside a rapid resumption of normative activities. Sleeping difficulties appear and are influenced by the child's age: 5–7-year-olds find it hard to settle to sleep; older children report nightmares, though some derive comfort from dreaming of the deceased [1]. Children of all ages settle more easily when an attachment figure is nearby. From the age of 8 years, physical manifestations of distress such as headaches appear, as do temper outbursts, argumentativeness and concentration difficulties. Children's natural curiosity about the death can sometimes reflect underlying anxieties about their 'responsibility' for what happened. Parental distress in response to their questioning can silence children, allowing misperceptions about the death to persist. Separation anxiety takes

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the form of worries about the vulnerability of loved ones.

Adolescence

Grief takes various forms, including withdrawing from family activities and/or seeking peer support. Adolescents may challenge their own mortality with risk-taking behaviour such as alcohol or drug use. Their cognitive ability to review their prior relationship with the deceased may bring comfort or, for those troubled by guilt or regrets, increased distress. They may acquire new family roles and responsibilities and encounter expectations of overmature behaviour. A sense of responsibility, alongside a desire to protect grieving adults, may result in disguised grief and mixed messages to others.

RESILIENCE AND POSITIVE OUTCOMES IN BEREAVED CHILDREN

The majority of children are resilient and follow a normal developmental trajectory subsequent to bereavement. Indeed, although parental death changes a child's life path irrevocably, some children and adolescents report positive responses following bereavement, including increasing independence, better school performance, heightened empathy to another's distress, and a growth in spirituality [3].

Research exploring positive bereavement outcomes indicates that resilient children show greater coping efficacy and fewer negative appraisals than affected children [4]. However, as 'resilient' children are chosen on the basis of being below a given threshold of clinical disturbance, differences in their cognitive styles may reflect differences in the mental health status of 'resilient' and 'affected' children.

PSYCHOPATHOLOGY IN BEREAVED CHILDREN

Reported differences in rates of psychopathology in bereaved children stem from differences in study inclusion/exclusion criteria, recruitment practices and measures used. The best controlled studies indicate that only one in five will show disturbance of clinical severity [5].

Commonly agreed bereavement symptoms include dysphoria (a state of unease), headaches, stomach aches and separation anxieties. Disturbance is generally non-specific with a marked heightening in the frequency and persistence of grief symptoms that in other bereaved children normally attenuate within 4 months of the death [3,5]. An expressed wish to be dead generally reflects the child's desire to be reunited with the deceased, although such statements require careful exploration particularly where family suicide has occurred.

Children bereaved by family murder or suicide can evidence post-traumatic stress disorder (PTSD) and internalizing disorders [6]. Rates and types of psychopathology are similar to those in other bereaved children [7], with a raised risk of depressive disorder up to 2 years after the event [8]. An increased risk of suicidal behaviour [9,10] and higher levels of persistent anger, guilt, shame and social isolation are also reported [7].

Complicated traumatic grief (CTG) is characterized by persistent intrusive and avoidant trauma symptoms that arise when the deceased died in subjectively traumatic circumstances. It can lead to avoidance of any positive or negative reminder of the deceased and social withdrawal at school [11]. The causes are unclear. One suggested contributory factor is that children's sense of predictability and stability can be undermined if their primary caregiver appears overwhelmed by the death [12]. Attempts to differentiate between disorders such as CTG and PTSD continue [13]. Short-term trauma-based cognitive behavioural interventions (CBT) with parents and children offer a promising approach to resolving CTG [11].

Recent research examines the hypothesis that the stress of traumatic parental death can lead to long-term dysregulation of the hypothalamic–pituitary–adrenal (HPA) axis in bereaved children [14]. The differences found between bereaved and control children in levels of cortical suppression, thought to reflect 'adrenal exhaustion' in bereaved children, were attributed to adaptation to chronic stress, leaving unclear how acute traumatic bereavement is associated with chronic HPA-axis dysregulation.

WHAT INFLUENCES CHILD OUTCOME?

Difficulties in obtaining representative samples of bereaved children make it difficult to examine systematically factors that moderate or mediate their outcome.

Child morbidity is influenced by the age and gender of the child. Younger children evidence behavioural or anxiety problems, while adolescents exhibit dysphoria or depression similar to that found in bereaved adults. Generally, boys exhibit higher rates of overall difficulties and acting out/aggressive behaviours than girls, who are more likely to show sleep disturbance, bedwetting and depressive symptoms.

Familial factors contribute to both child morbidity and resilience. Parents who report having mental health difficulties post-bereavement are more likely to have children with higher rates of child disorder [5]. The converse appears true for 'resilient' children, whose parents have lower levels of disorder than do those of affected children [4]. Child resilience is promoted by parental warmth, authoritative parenting and consistent discipline [4,15]. More recently, self-reports of interpersonal loss and conflict, and greater fears of abandonment have been linked to internalizing symptoms in bereaved girls [16].

Pre-bereavement factors predisposing to post-bereavement child disturbance include mental health difficulties, marital conflict or separation, though this information rests on the retrospective recall of distressed parents. Genetic factors may influence child outcome in families where members have pre-existing psychiatric disorders.

INTERVENTIONS WITH BEREAVED CHILDREN

Theoretical and cultural influences

There are two main theoretical bases for interventions with bereaved children. The first suggests that children need to complete a sequence of bereavement-related tasks in order satisfactorily to resolve their grief and avoid maladaptive outcomes. These tasks include acceptance of the permanence of loss, constructing a positive internal image of the deceased, and finding new and supportive relationships [17]. In CTG, trauma is conceptualized as interfering with grief resolution, necessitating the relief of traumatic symptomatology so that grieving tasks can be accomplished [11].

In the second approach, children's outcomes are seen to be the product of multiple, cumulative risk and protective factors operating in the post-death environment. Interventions within this framework aim to foster resilience, for example by moderating children's coping styles and supporting positive parenting [18].

Cultures provide frameworks that guide beliefs about death, define 'positive' and 'negative' outcomes, and govern the expression of grief. For instance, in some cultures expressions of sorrow bring social opprobrium. Therapists need to gain an understanding of children's familial, cultural and ethnic backgrounds, and be aware of how their practice is influenced by Western conceptualizations of grief and mourning.

What do bereaved children need?

Bereaved children benefit from receiving accurate information about the death and related events. Reassurance that they could not have changed or influenced either, and that death mostly affects the elderly, is helpful. Explanations need to be age appropriate, clear and truthful, avoiding euphemisms such as 'gone to sleep', which young children will interpret literally. Little guidance is offered where familial suicide or murder occurs. Open sharing of information may be affected by protectiveness towards children, or by adult survivors' guilt and shame [10]. Adults may struggle with conveying understanding and empathy for the deceased without suggesting to vulnerable children that violence or suicide are acceptable coping strategies [7].

Children benefit from the re-establishment of consistent daily routines, the emotional availability of major caregivers and having their developmental competencies appreciated and fostered. Engagement in the wider social world, via activities and friendships, should be maintained.

Children are also helped by involvement in family expressions of grief, such as choosing flowers for the deceased or speaking at the funeral service. They report that physical comforting and sharing their thoughts and feelings within the family are helpful. Recognizing, normalizing and discussing their grief and concerns provides all children with a sense that death can be managed and need not overwhelm them.

What is helpful for parents?

Distressed parents can, understandably, be uncertain about what to tell bereaved children and when. Often all that parents require is an opportunity to discuss their concerns and potential responses with an understanding and supportive adult, who can offer advice on management. Unexpected deaths require quick decisions from unprepared parents, who can be reassured that decisions they later regret can be addressed subsequently. For example, children who did not attend the funeral service can have it described to them, and they can visit the burial site. Explanations that children's grief can take different forms and will attenuate over time can reassure parents about the normality of their children's responses. Family reminiscing about the deceased is of particular help to children who appear not to be grieving [1]. Giving children mementos of their dead parent or sibling provides them with comfort and helps them to maintain a positive relationship with the deceased. Normal limit setting increases children's sense of security. Schools can promote bereaved children's resilience by providing understanding and support and by incorporating preparation for trauma and loss into educational thinking and practice.

Services for bereaved children

There has been a notable expansion in community-based services for bereaved children. Yet, quantitative evaluations of controlled bereavement interventions have highlighted few positive treatment effects [19,20]. Treatment effects may be limited by the fact that it is not necessarily the level of child distress that determines who receives services [5], and that outcomes judged in terms of changes in psychopathology may be ill-matched to therapeutic inputs [19]. Interventions are neither neutral nor always helpful and infrequently measure potentially negative outcomes, such as an increase in child distress.

The role of professionals

The majority of bereaved children need neither professional intervention nor therapy. Referral is appropriate where there is prolonged distress or disorder (Table 16.1). It is essential to gain a detailed understanding of the circumstances of the death, what information and explanations the child has been given and how their knowledge was acquired. Relevant cultural or religious variables need to be understood.

It is therapeutically useful to see children and parents separately and together. Individual meetings with children can highlight hidden worries, cognitive distortions, self-blame or symptoms of trauma. Individual meetings with parents can highlight concerns about what information to share with children. Seeing family members together can highlight which family processes to strengthen or modify. Widening family support networks is useful – for instance, via school consultations or reassuring parents that other trusted family members or friends can also help in managing children's grief.

Cognitive behavioural therapy (CBT) with children and their parents can effect significant improvements in PTSD and internalizing disorders and complicated grief [12].

CONCLUSIONS

Bereaved children's expression of grief is influenced by their developmental level and their age and gender. The episodic quality of their grief can be confusing to adults. Separation from attachment figures can induce anxiety across all age groups. Grief-related distress does not indicate pathology. Clinical disturbance affects approximately one in five children. Positive and authoritative parenting facilitates childhood resilience. Parents appreciate information that normalizes children's grief and traumatic symptoms, and provides guidance on management. Bereaved children value opportunities to share their thoughts and feelings. The majority of families do not require psychological services, although families bereaved by murder or suicide would benefit from extra support. Consultation with other agencies in routine contact with children, such as schools, can enhance families' supportive community networks.

Referral for professional help is appropriate when grief and trauma symptoms disrupt normal functioning and persist over time. It is essential to consider the impact of religious and cultural beliefs upon presentation and the implications of these for appropriate therapeutic goals and practice.

Table 16.1 Bereaved children: assessing the need for support and intervention.

Time	Context	Post-death		
	Implications for Management	**Monitor**	**Advise/consult with professional**	**Consider referral if:**
Pre-death	*Children*:			
	Developmental level	If learning disabilities	Liaise with school	
	Understanding of death			
	Temperament			
	Relationship with deceased	If problematic		
	Prior history of loss/divorce/ separations/death	Yes		
	Prior disturbance/disorder	Yes		
	Family:			
	Patterns of communication			
	Organization			
	Role differentiation			
	Quality of family relationships	If poor/prior disturbance		
	Prior parental mental health difficulties	Yes		
	Culture/religion/community:			
	Beliefs			
	— meaning of life/death/ afterlife — relationships with the dead — roles of adults/children/ family			
	Behaviour			
	— culturally appropriate rites/ rituals — culturally appropriate expressions of grief	Practical obstacles/ community support	Consider support from/liaison with cultural community	
Death occurs	*Nature/circumstances of death*:			
	Traumatic, murder, suicide	Yes		
	Presence of child			
	Information provided			
	Adults available to child			
	Degree of preparation/ support available			

Table 16.1 (*continued*)

Time	Context	Post-death		
	Implications for Management	**Monitor**	**Advise/consult with professional**	**Consider referral if:**
Immediate aftermath	*Children*:			
	Impact upon routines/practical care			
	Suitability of substitute care	Yes	If unsuitable over longer term	
	Involvement in rites/rituals		If requested	
	Expression of grief tolerated			
	Opportunities to gain understanding			
	Family:			
	Explanations given to child		Psycho-education on grief if requested	
	Availability of practical/emotional support for all family members			
	Support for expression of grief and distress			
	Extent to which able to appreciate children's needs			
	Extended family: involvement with child			
	conflict around death/ aftermath	Yes		
Short term (up to 4 months)	*Children*:			
	Degree routines/activities re-established	Absence from school	If prolonged — family/school liaison	Persistent
	Ability to use support within family/from peers			
	Type of grief symptoms	If signs of trauma	Psycho-education — monitor	Severe/ persisting
	Opportunities to express grief			
	Level of distress/avoidance	If marked	Psycho-education — monitor	
	Opportunities to consolidate, increase understanding			
	Lack of stability/further losses	Yes	Family/school liaison — monitor	
	Family:			
	high level of distress/ mental health difficulties		Assess help needed/ wanted	Interfering with functioning
	Lack of support for adults		Consider ways of increasing — monitor	

(*continued overleaf*)

Table 16.1 (*continued*)

Time	Context	Post-death		
	Implications for Management	**Monitor**	**Advise/consult with professional**	**Consider referral if:**
Longer term	*Children*:			
	Persistence of marked distress/ emergence of disturbance		Psycho-education/ increase support	Interfering with functioning
	Trauma symptoms			Yes
	Difficulties with peers		School liaison	Persistent
	Family:			
	Adult mental health difficulties		Assess help needed/ wanted	Persistent
	Marked relationship difficulties		Assess help needed/ wanted	Persistent

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17

Adoption and Fostering

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There are now very few non-relative adoptions of UK infants, and international adoptions have declined in the UK as elsewhere since 2004 [1]. Most UK adoptions are of older 'looked after' children, who have usually been fostered prior to adoption.

CHILDREN ADOPTED IN INFANCY

These adoptees show somewhat higher levels of difficulty than non-adopted children, mainly disruptive problems [2,3], with difficulties most marked in later childhood and early adolescence [4]. Although the great majority of infant-adopted children are within the normal range of adjustment, clinical referrals of infant-adopted children are substantially higher than for non-adoptees, apparently reflecting a small high-risk subgroup [5,6].

Genetic risks probably account for some of this over-representation in clinical populations. However, a well-functioning adoptive family environment acts as a developmental protective factor for children whose biological parents had a heritable mental illness, abused alcohol or had a criminal record [7].

Early short-term interventions can increase parental sensitivity and infant attachment security, and reduce rates of attachment disorganization, in infant adoptions [8,9]. After infancy, clinically important issues concern children's developmentally changing understanding of adoption [10], and curiosity about their origins. Children need to revisit these questions as they mature cognitively and emotionally. Where adoptive parents are uneasy with the subject, children may avoid raising it; so parents may believe, sometimes with relief, that the child is 'not interested' or 'already knows everything'. Voiced or not, children's questions focus around two areas. One is the birth parents – who were they? what were they like? Adopters need good information here, to share over time, and need to help the child to feel that their adopters value aspects of them derived from birth parents as well as from their adoptive upbringing. Where information is lacking, as in many international adoptions, children still need mental representations of their birth parents; parents can help them appreciate that their own characteristics may offer clues. The second focus of children's questions is why they were given up. Commonly, young children have an underlying feeling that something was wrong with them that led the birth parents to reject them. With age children become more able to understand the complex reasons why birth parents may have been unable to care for them.

We now turn to foster children, and then to the small subgroup who move on to adoption.

FOSTER CHILDREN

The great majority of looked-after children (LAC) in the UK are fostered. Foster carers often possess little information about the child's history with which to make sense of their behaviour. Even in stable, long-term foster families, children may lack a sense of permanency or belonging. LAC show

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markedly lower educational performance than other children, with the gap widening at each stage. Children entering care are already a severely socially disadvantaged group, but foster care does not remedy this disadvantage [11]. Earlier maltreatment profoundly affects educational attainment among foster children, even in stable placements [12]. Adopters can often advocate strongly for their children's needs, in contrast to many foster carers, who lack the adopters' legal status as parents, their lifetime commitment to the child, and often their educational and social background.

Looked-after children show much higher rates of psychiatric difficulty than children remaining in their families. Ford *et al*. [13] found that 46.4% of LAC had at least one ICD-10 (*ICD-10 International Classification of Mental and Behavioural Disorders in Children and Adolescents*) psychiatric diagnosis, compared to 14.6% of deprived and 8.5% of non-deprived children living in private households. Children in residential care were most disturbed, but 38.6% of foster children showed psychiatric difficulties. Comorbidity was high, an issue discussed below in relation to psychiatric classification. Few children entered care because of any parental illness, so psychiatric disorder in the birth parents cannot account for the enormous discrepancy between LAC and others. Although experiences in care may themselves be associated with difficulties (e.g. prevalence of disorder was higher where there were many recent placement changes) the children's psychiatric difficulties are likely to derive mainly from abusive or neglectful parenting. In addition, neurological changes have been described in children subjected to prenatal and neonatal stress, likely to be more common in socially disadvantaged families; and neurobiological changes are also known to result from early maltreatment [14,15].

Besides higher rates of difficulty on measures designed for community and ordinary clinic populations, maltreated children also show complex and clinically significant problems that are not well captured by these measures [16]. A recently developed measure designed to examine such difficulties is the Assessment Checklist for Children (ACC). The ACC scales illustrate the kinds of problem very commonly encountered in clinical work with maltreated children, including very disturbed relationship styles, self-injurious behaviours, disturbances around eating, responses to trauma, and inappropriate sexual behavior [17].

Current DSM-IV-TR (*Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition – Text Revision*) psychiatric diagnostic classification does not adequately capture the difficulties shown by the population of maltreated children. Children may receive multiple diagnoses, not linked by reference to the child's developmental history and maltreatment experiences; or show subthreshold difficulties in numerous areas, causing real impairment not reflected in their diagnosis.

Maltreatment is unlike other forms of trauma in several ways. It usually occurs early in development; is chronic rather than a discrete event; and takes place within the child's attachment relationship, so that parental figures who would be expected to provide protection are themselves sources of fear (producing attachment disorganization). It is often cumulative, involving several different types of trauma, and this is associated with symptom complexity [18]. Certain forms of maltreatment may not meet the trauma criterion of involving threat of death or injury, although they may still produce symptoms of post-traumatic stress disorder (PTSD).

It has been argued that the resulting psychological damage to children is better captured by a diagnostic category such as childhood complex trauma or developmental trauma disorder [19], reflecting the derailment of normal developmental processes across multiple domains and the organization of behaviour to prevent recurrence of trauma effects. Instead of a child receiving several apparently unrelated diagnoses – e.g. attention deficit hyperactivity disorder (ADHD), conduct disorder, reactive attachment disorder and separation anxiety – such a diagnostic category would recognize a pattern of coexisting and somewhat interrelated difficulties across several domains in maltreated children. These include attachment, emotional dysregulation, behavioural and impulse control, attention and cognition, dissociation and somatic dysregulation.

Children adopted from care

While most LAC are aged 10 years or older, the majority of children adopted are under 5 years of age. It should be noted that Ford *et al*. [13] found that risk of psychiatric difficulties was just as high in this younger age group as in older children.

Two obvious areas of difficulty in later-adopted and foster children are the impact of maltreatment, and difficulties in attachment and relationships (see Chapters 15, 18 and 19).

Regarding maltreatment, the ACC, mentioned above, assesses several areas of likely difficulty. Clinicians should be alert to possible post-traumatic symptoms, and depression, often comorbid with PTSD. Traumatic memories may only resurface after the child feels safe in foster care, or after the permanency of adoption. Triggers may include later losses or severe stresses as well as reminders like anniversaries or places. Children may have only fragmentary memories, and may feel 'crazy' or overwhelmed by emotions, flashbacks or dreams, and reassurance is essential. The original traumatic events have often not been known to services or foster/adoptive parents, making it more difficult for adults to help the child make sense of traumatic memories or feelings.

Regarding attachments, maltreated children are obviously at high risk of an 'insecure' attachment organization; but additionally, because the attachment figure whom they need as a source of security is simultaneously a source of fear, they are at greatly increased risk of attachment disorganization [20]. Insecure-disorganized attachment, much more than insecure attachment alone, is related to later behavioural and emotional difficulties, including aggressive and oppositional behaviour, later dissociative symptoms, and poorer self-confidence and social competence [21].

Once these children enter adoptive or foster families, they must form new attachments with strangers, much later than normal and on the basis of existing internal working models of attachment, which can profoundly affect their expectations of new parental relationships. Attachment difficulties often relate to other areas of behaviour and can perpetuate existing models. For example, children may avoid showing a need for comfort or affection, so as not to reveal (or feel) dependence or vulnerability. This may have been the best available strategy for the child who could not expect comfort, but may conceal from new adoptive parents the chance to respond in a way that could begin to alter the child's expectation.

Children showing difficulties in their relationships with caregivers (usually alongside other behavioural and emotional difficulties), are often described as showing an attachment disorder. 'Attachment disorders', as defined by ICD-10 and DSM-IV, cover two types of difficulty, the precondition of both being very adverse early caregiving. Not all maltreated children show such disorders, and some children show features of both. These types are:

- Directing sociable and attachment behaviours towards people without showing the usual selectivity ('disinhibited'). Such indiscriminate behaviour appears fairly resistant to change, even though the child may also begin to show clear attachment behaviour towards a preferred adult once long enough in placement.
- Inhibition of sociability and of seeking and accepting comfort ('inhibited'); this usually changes once the child has a responsive caregiver.

Alongside these defined classifications there has also been an explosion in the use of the term 'attachment disorder', claimed to underlie a vast range of difficulties. Many popular websites put forward a version that 'is not discernibly related to attachment theory, is based on no sound empirical evidence and has given rise to interventions whose effectiveness is not proven and may be harmful' [21]. 'Attachment disorder' as a diagnosis for older maltreated children should not be overextended to their difficulties in other areas of functioning, which need examination and treatment in their own right.

TREATMENT CONSIDERATIONS

Where children have suffered maltreatment and disruption before adoption placement, they can profoundly affect previously well-functioning adoptive families, and clinicians should be wary of pathologizing these families as the apparent source of difficulties. However, models that locate all the difficulty in the child's behaviour and abuse history, seeing adoptive parents as 'co-therapists', risk denying the importance of the child–parent interaction; abused children can 'push the buttons' of particular vulnerabilities in adoptive parents, in ways that are not necessarily predictable either by professionals or parents.

An important part of clinical work is a history of the placement, including the adoptive parents' expectations, what information about the child they were actually given, what potential difficulties

they were led to expect, whether they can identify likeable qualities in the child, and what support they have, including extended family.

Adoptive parents need full background information about the child's history. If this is lacking it should be obtained as soon as possible; the clinician can use this with the parents to help them attune and make sense of their child's responses, and alter negative interaction patterns. The child needs help to construct a coherent story of their life if one is lacking. This is often done in the form of a 'Life Story Book', which should incorporate the child's own memories and feelings alongside a chronological account. Parents should be fully involved in this, rather than it being seen as direct individual work with the child; this helps child and parents to share the child's history, helps in understanding the child better, and helps adopters towards the role that parents ordinarily fulfil in relation to their birth children, of a 'memory bank' that the child can draw on when needed.

As regards attachment, the move to new adoptive parents is itself the most radical form of treatment possible. A study assessing the child's attachment representations showed increasing security over the first 2 years of placement, although insecure and disorganized representations also persisted [22]. Reports of adoptive parents and retrospective reports of adopted adults indicate positive results for the majority of late-adopted children in terms of attachments and relationships, and show that even where adolescence is very troubled, improved family relationships may follow. Support for parents is essential; sensitive caregiver interaction with the child, and the capacity to respond in security-promoting ways even to negative and provocative behaviour by the child, can be difficult for parents to maintain.

As the developmental trauma of maltreatment has usually occurred in the context of the child's attachment relationship, treatment approaches often incorporate work on both, although techniques such as trauma-focused cognitive–behavioural therapy (CBT), or Eye movement desensitization and reprocessing (EMDR) may be useful where there are particular traumatic incidents and PTSD symptoms. The 'Attachment, Self-Regulation and Competency' (ARC) framework for treating complex trauma [23] focuses on increasing positive attachment, helping the caregiver to manage the child's affect, attune to the child, respond consistently and develop safe, predictable routines. On this basis, the ARC framework outlines interventions designed to develop other competencies damaged by the history of maltreatment, such as the identification and regulation of emotion, including psycho-education about the trauma response; cognitive competencies, including executive function skills; and social skills.

Numerous therapeutic approaches to attachment difficulties have been described, but systematic evaluation is lacking. Examples of interventions are given in Box 17.1.

There is no evidence for benefit from 'holding', 'rebirthing' and similar 'attachment therapy' techniques that employ physical restraint or

Box 17.1 Examples of interventions for attachment difficulties

- Direct work to support parents, e.g. in helping them to reframe behaviour via knowledge of the effects of maltreatment, and hence to manage it differently. A 'story stem' narrative assessment of the child can give a picture of the child's expectations and perceptions of attachment relationships, which can help parents attune better to a child.
- Standard parenting programmes, e.g. Webster–Stratton, adapted to include adoption-specific areas.
- Active learning in the child–parent interaction, with the therapist supporting the parent's responses, and directing and facilitating the child [24].
- 'Theraplay' [25] involving active engagement of the child in physical play with the therapist, seen as modelled on the healthy parent–infant relationship, with the parent involved first as observer and later as co-therapist.
- Individual psychoanalytic psychotherapy for the child, with concurrent work with the parents, can help to alter underlying negative expectations of attachments and relationships [26].

domination, coercion, regression, and so on. These are not based on attachment theory, though usually claiming to be so; they risk retraumatizing a child already traumatized by an adult in a parental or 'caring' role; they have been responsible for a number of child deaths and are strongly contraindicated [27].

Parental satisfaction with adoption is generally high, despite difficulties. Adoption breakdown rates vary greatly between agencies, attesting to the importance of preparation and post-adoption support for the family. Parents often feel that Child and Adolescent Mental Health Services do not adequately understand these children's difficulties; as described above, problems are inadequately captured by the usual diagnostic classifications, and treatment provision may be fragmented if organized by diagnosis. Support is essential, but its availability varies enormously. Local authorities now have a duty to provide assessment and support services (often outsourced to other agencies), but variation is likely to continue in the level of services provided.

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Section 4

The Impact of Trauma and Maltreatment

18

Stress and Reactions to Stress in Children

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WHAT'S NEW

- Stress in pregnant mothers can be transmitted to the foetus and cause enduring effects on offspring [14].
- Diagnostic criteria are being revised to allow more rational diagnosis of complex trauma in children [11,17].
- Building resilience is recognized as a key factor in treatment of stress-related disorder [27].
- Trauma-focused psychological therapy has been shown to be more effective than medication in the treatment of PTSD [24].

WHAT IS STRESS?

Stress may be defined as a real or interpreted threat to the physiological or psychological integrity of an individual that results in physiological and/or behavioural responses [1]. It is increasingly recognized that stress cannot be fully defined in objective terms. The way in which an event is *perceived* as stressful or threatening has a major effect on the way an individual will respond and is influenced by:

- the child's developmental stage;
- the circumstances surrounding and following the incident;
- the support subsequently available.

THE PSYCHOPHYSIOLOGICAL RESPONSE TO STRESS

The ability to perceive danger and to protect ourselves effectively from it is crucial for our day-to-day survival. Danger is a powerful stressor and produces an automatic psychophysiological response enabling us to 'freeze, fight or flee' [2]. When danger is perceived, there is an immediate arousal response via the limbic system (Figure 18.1), with activation of the hypothalamo–pituitary–adrenal (HPA) axis and inhibition of non-essential physical and psychological functions. The body is prepared for action. Sensations become more acute, attention becomes highly focused towards the potential danger and reaction time speeds up. Pain sensation may be diminished, as a consequence of the release of endorphins in the brain, even after severe injury. The release of noradrenaline and cortisol into the bloodstream helps to ensure that the body remains able to cope with stress for a considerable period of time. Memory processing and other cognitive functions are also affected [3,4]. In extremis, a freeze response can occur, when pulse and respiration slow and may even stop [5]. Of course, not all stressful events will be experienced as dangerous and the associated stress response is likely to be modulated accordingly.

Later reactions after a stressful event

When safety and security are felt to be restored, recovery can be rapid; but where the situation involves dealing with ongoing stress, different types of response will occur. For example, loss of a

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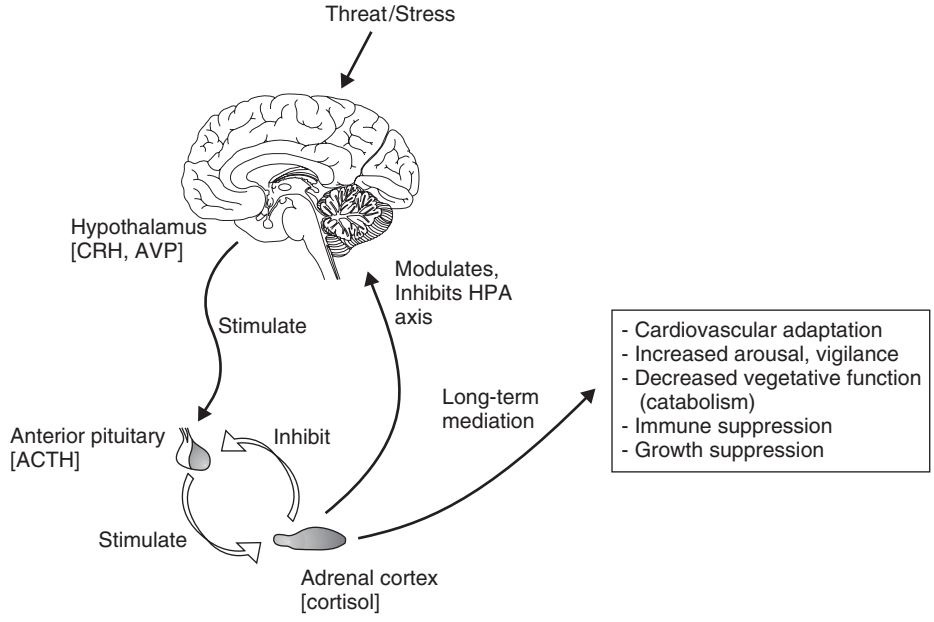

Figure 18.1 The hypothalamic–pituitary axis (HPA) and the threat response. ACTH, adrenocorticotropic hormone; AVP, arginine vasopressin; CRH, corticotropin-releasing hormone.

loved one by death or separation may be followed by a bereavement response in which initial numbness gives way to grief and mourning. When loss is unexpected and traumatic, however, the initial shock may be followed by intrusive thoughts and images that make it difficult to come to terms with the loss in the normal way. This may give rise to complicated and prolonged grief reactions.

Some stressful events bring with them enormous changes for the individuals affected. Natural disasters or war, for example, disrupt the way of life of whole communities for long periods. But even changes on a smaller scale can result in catastrophic disruption of the individual's assumptions about the world, including the framework for self-identity, understanding events, planning and taking action. Repairing the damage and developing new ways of functioning may take considerable time [6].

Longer-term effects

Much of what is known about stress and reactions to it comes from research in the USA with Vietnam veterans. This has shown that the effects of extreme, protracted and repeated stress can be enduring. Mental health problems range from relatively mild and self-limiting to severe and long-lasting. Although they are necessary for survival, it seems that frequent neurobiological stress responses increase the risk of physical and mental health problems, particularly when experienced during periods of rapid brain development [7,8].

DEVELOPMENTAL ISSUES

Our bodies and brains automatically respond to dangerous situations without understanding or conscious awareness. For example, in a newborn any sudden change of sensation is sufficient to provoke a startle response. As young children grow older, what they learn about danger and safety lays the foundations for how they will understand and cope with stress in later life. The relationship between the child and its primary caregiver provides a particularly crucial learning environment. If this environment does not provide protection but instead exposes the child to repeated danger (e.g. domestic violence or abuse), there are likely to be long-term effects both on the child's assumptions about the world and on their mental health [8,9].

EPIDEMIOLOGY AND HISTORY

The effect of stressful experience will depend on an individual's ability to cope and the support that is available, as well as on the event itself [10]. There appears to be a range of possible stress-related disorders, ranging from mild to severe (Box 18.1).

At present, stressful experience is only formally required in diagnostic criteria for adjustment disorders and post-traumatic stress disorder (PTSD). The conceptualization and understanding of stress response syndromes has changed enormously over the last century and remains a major focus for research. For example, it was only in the 1980s that criteria for PTSD were published in DSM-III (*Diagnostic and Statistical Manual of Mental Disorders*, 3rd edn.), with the ICD-10 (*ICD-10 International Classification of Mental and Behavioural Disorders in Children and Adolescents*) definition following in 1992. Further revisions are expected with the publication of DSM-5 in 2013 [11,12].

The experience of stress is part of normal life, and traumatic stress is far from rare, especially in populations afflicted by violence or natural disaster. The prevalence of mental health disorders resulting from traumatic stress can only be guessed at, but are clearly considerable. For example, epidemiological studies of PTSD alone estimate a point prevalence of 1%, and 4–12% for a lifetime diagnosis [13]. Rates in clinical populations, however, are likely to be much higher.

Box 18.1 The response to stress

Stressful experiences can lead to:

- Normal stress responses
- Adjustment disorders
- Psychiatric disorders, including:
  - mood disorders (anxiety states, PTSD, depression)
  - dissociative states
  - psychosomatic complaints
  - eating disorders
  - attachment disorders
  - personality disorders in adulthood
  - substance abuse.

STRESS REACTIONS IN CHILDREN

A child's immediate reaction to extreme stress is usually one of distress and tearfulness. Once the event is over, it takes time for the child to adjust to and recover from what has happened. The nature and severity of the reaction will depend on characteristics of the event, factors affecting the child's resilience, and the recovery environment [14,15]. The type of stressful experience, the suddenness of the event, the amount of preparation that has been possible, the sensory exposure entailed and the degree of secondary trauma will all affect the impact of the event on the child. The appraisal of the stressful event – what it means for the child and how this is processed cognitively – is one of the most important factors in determining how an individual responds and copes.

Stressful experience that overwhelms the child's coping abilities can be traumatizing even when not actually life-threatening. Young children who experience overwhelming sensory exposure during the event may be unable to process this cognitively, which makes them particularly vulnerable to flashbacks and intrusive re-experiencing of the event (Box 18.2).

Box 18.2 Factors affecting risk and resilience

- Type and duration of traumatic experience
- Perceived severity of stress/trauma exposure
- The child's age and maturity
- Exposure to stress at a young age
- The child's gender
- Personality characteristics
- Previous exposure to stressful experience
- Time elapsed since exposure (symptoms often reduce over time)
- Pre-trauma psychopathology
- Coping abilities/resources
- Parental mental health problems
- Social/cultural resources and support

If the consequences of the event are manageable and disruption is minimal, the child's distress and upset may resolve within a few days or weeks. The ability of parents (who may themselves be distressed and traumatized) to provide adequate care, and the presence of a stable caring system around the child, is crucial in promoting recovery. When these are absent, significant mental health problems can develop. And of course, if traumatic experience is repeated there is a risk that chronic and complex responses will develop [16].

PSYCHOPATHOLOGY

Following a traumatically stressful experience, repetitive and intrusive memories of the event are common. A chance noise or other stimulus may trigger a flashback. Disturbing images may also occur at quiet moments such as bedtime, making it difficult for the child to settle to sleep. In very young children, sleep disturbances such as night terrors and night waking are common. Older children often report that their sleep is disturbed by bad dreams and nightmares. Children who are too young to be able to understand what has happened to them or to express their thoughts and feelings in words are likely to re-enact a traumatic event in their play or drawings. They often develop symptoms of hyperarousal, such as overactivity, irritability, difficulty concentrating and hypervigilance. Signs of generalized anxiety are also common, including clinginess and fears of the dark. Language, toilet-training and other developmental skills may regress or even be lost altogether. The child may become withdrawn or unresponsive and try to avoid situations, objects or even words that remind them of the traumatic experience.

When children are exposed to chronic and repeated stress such as abuse or domestic violence, many domains of development may be affected [17]. Such children present a diagnostic challenge to the clinician, not uncommonly presenting with complex disorders of arousal, mood and conduct. When trauma is not identified, such children may be misdiagnosed as suffering from attention deficit hyperactivity disorder (ADHD), conduct disorder or psychosis.

As children get older, their reactions to extreme stress become more like those of adults. Adolescents may meet DSM or ICD criteria for PTSD and other disorders. Those exposed to prolonged or repeated stress may also present with dissociative symptoms, angry outbursts, self-injury and substance abuse [16]. Memory and concentration difficulties are common and can affect school work and grades. Moodiness, anxiety, depression and irritability can put pressure on peer and family relationships.

ASSESSMENT

When asked to assess a child following a major stressful event, it may be helpful, before arranging to meet the child, to have a planning meeting with the parents/carers. Interviewing parents and child together about the child's current circumstances and functioning is often a good place to start the assessment. Parents can provide useful background information such as family history, and the child's developmental history. It is often helpful to see parents separately to obtain information about the parental developmental history (including trauma and attachments), marital relationship, and life experience of separations, abuse, illness and other stressors or life events. The parental account of any traumatic events should include their own and the child's reactions to the trauma, and how these have been managed.

Interviewing the child individually is especially important following traumatic experience. Parents often lack crucial detailed information about their child's experience and may therefore be unable to provide all the information needed. Children often communicate more freely about their experiences when not afraid of causing distress to carers. It is usually necessary to ask the child direct questions about their experience of traumatic events and their symptoms. Asking the child about what has helped them to cope is also important. A semi-structured interview can be helpful [18] (Box 18.3).

Young children respond best to an approach using play and drawing to help them to express themselves. At the end of any interview about trauma, the interviewer needs to help the child 'wind down', to review and summarize the session, and to discuss anything that was particularly disturbing or helpful. Providing information about traumatic experience and its consequences helps to

Box 18.3 Trauma interview for children and adolescents

1. Ask the child:
   - 'Have you ever been in [e.g.] a car accident ... or a house fire [other stressful experience] ... or thought you might get hurt or die?'
   - 'Have you ever seen someone else get hurt badly?'
     'Did someone important to you ever die, such as someone in your family or a good friend?'
2. Having identified traumatic event/s, ask the child to tell his/her own story about it/them. Use free recall as much as possible.
   - Assist recall with questions about context of the traumatic event (e.g. when/where/who.
   - Ask about the child's physical/psychological responses at the time.
3. Track the course of symptoms from immediately after trauma to the present.
   - Ask about meaning/attributions: the child's feelings (e.g. of guilt and being different, damaged or isolated); reactions of others.
   - Ask about what has helped them to cope.
4. Ask the child to describe his/her thoughts/plans about the future.
5. Wind down (see text).

normalize the child's reactions; acknowledging the child's courage in having shared their experience can boost self-esteem.

DIFFERENTIAL DIAGNOSIS

Stressful experience can provoke a range of possible responses, both normal stress responses and a variety of post-traumatic stress disorders [19]. A single, brief, unexpected stressor (e.g. an accident) is likely to produce a very different response from traumatic experiences that are repeated, prolonged and expected (e.g. sexual abuse). Depending on the circumstances, a child may develop the symptoms of a disorder (e.g. PTSD) but fall short of meeting the full criteria. The most common disorders are listed in Box 18.1. Comorbidity is common following severe trauma [12].

Careful history-taking and clinical examination are crucial for accurate diagnosis. Traumatized children are often withdrawn, avoidant or dissociative. This affects their ability to communicate, so that definitive diagnosis is likely to take time. Standard questionnaires can be helpful adjuncts to the detailed clinical interview [20] and provide useful baselines for treatment.

EFFECTIVE MANAGEMENT AND TREATMENT

Management will depend on the specific circumstances and needs of the individual child and family. A broad, multisystemic approach may be needed in order to identify and address all of the child's needs, especially following chronic or complex trauma [16]. Situational factors such as family adjustment problems, school difficulties or complex legal processes could cause significant ongoing stress and require practical help or advice. Clearly, given the range of possible stress-related disorders, a number of treatment approaches may be needed. What follows is just a brief outline of what may help a child to recover from severe stress.

Immediate measures

For any child who has been exposed to life-threatening danger, the most immediate requirement is restoration of safety and security. Much distress in the immediate aftermath can be alleviated by providing basic information and practical help. Later, screening to identify those at risk of developing mental health problems can also be valuable [13].

Psychological First Aid is an early intervention that aims to promote healthy recovery from traumatic stress by ensuring that support can be accessed, thus optimizing the ability to cope and enhancing resilience. An approach known as Psychological Debriefing remains controversial owing to conflicting reports about its efficacy [21].

Cognitive-behavioural treatment (CBT)

This well-validated approach essentially relies on helping the child to recall the distressing experience (imaginal exposure) in such a way as to reduce distress (desensitization) and enable symptoms to be mastered (cognitive restructuring) [13]. Very young children, however, are unlikely to be able to make use of formal CBT, but can benefit from similar therapeutic approaches using play, drawing and narrative techniques.

Eye movement desensitization and reprocessing (EMDR)

This is a relatively new technique that has shown promising results so far in research with traumatized adults [22–24]. As yet, there are few controlled trials with children [13]. Symptom improvement is rapid and well maintained, even in very young children. EMDR uses many of the same elements as CBT but relies less on homework and verbal competency. It is particularly helpful with avoidant, or very young children.

Medication

Medication is increasingly used as an adjunct to multidimensional psychological therapy packages for post-traumatic disorders [25]. However, few studies have looked specifically at children [13]. Antidepressants, especially selective serotonin reuptake inhibitors (SSRIs), are effective, at least in the short term, in treating symptoms of hyperarousal, such as irritability and sleep disturbance, as well as those of depression. Night terrors, startle responses, avoidance reactions and overactivity may respond to propranolol (a β-blocker) or clonidine (an α_2-noradrenergic agonist) [26].

Box 18.4 Practice points

- Traumatic experiences are common and can have long-lasting psychological effects [13]
- Children are especially vulnerable to the effects of extreme stress [8,9]
- Parental accounts are unreliable – it is therefore important to interview the child
- Specific treatments work best as part of a multi-modal intervention [13]

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19

Child Maltreatment

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INTRODUCTION

Child abuse and neglect is a relatively common experience in childhood. At the very least it is unpleasant, and at worst fatal. Non-fatal child abuse and neglect causes a variety of harmful effects that are mostly psychological and behavioural, though some are physical. Intention to harm children is not required for the definition of child abuse and neglect. Different forms of maltreatment are recognized (Table 19.1) and they often co-occur. Retrospective studies of adults suggest that different forms of abuse and neglect lead to different sequelae [1] but because of the co-occurrence of different forms of abuse, it is difficult definitively to apportion the nature of the harm to the different forms. Nevertheless, some more robust associations are now recognized, as outlined below in the section 'Harm to the child'.

TYPES OF MALTREATMENT

Table 19.1 shows the four types of maltreatment. Most cases of maltreatment occur within the family [2], with children being harmed either by their parents or primary carers, and occasionally by siblings. The exception to this is sexual abuse, which is equally commonly perpetrated by someone who is known to the child or young person, but is not a parent or sibling. Child maltreatment is recognized across all cultures [2]. Some cultural practices, such as female genital mutilation, constitute maltreatment.

While physical and sexual abuse may consist of single or repeated events in the child's life, emotional abuse and physical neglect are more appropriately considered as pervasive aspects of the primary carer–child relationship. Table 19.2 lists contrasting features and relationships of the different forms of child maltreatment.

EPIDEMIOLOGY

In England in 2009, 34 100 children were subject to a Local Authority child protection plan, resulting from a decision based on a multidisciplinary consensus that the child *continues* to be at risk of harm, rather than on the substantiation of maltreatment. These numbers are, therefore, an underestimate of the actual prevalence of child maltreatment. Table 19.1 shows the distribution of forms of maltreatment within all child protection plans in 2009 in England. However, epidemiological findings depend on the source of the data. Self-reports by community samples of both children and, retrospectively, adults indicate that official statistics from child protection agencies present a substantial, up to tenfold, underestimate of the occurrence of the various forms of abuse [3].

SOCIAL AND FAMILY FACTORS

Physical child abuse and neglect are more clearly associated with social disadvantage in the families of the children [4]. People who abuse children, including parents, are troubled individuals, a proportion of whom have experienced abuse or neglect in their own childhood. Adolescent boys who sexually abuse children are more likely to

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Table 19.1 Forms of maltreatment, distribution within protection plans and cumulative rates based on self-reports, for England in 2009.

Type of maltreatment	Variants within type of maltreatment	Percent of child protection plans (2009)	Percent of young adult respondents reporting childhood maltreatment [3]
Neglect		46	6—12
	Lack of provision		
	Lack of supervision		
Physical abuse (non-accidental injury)	Causing death, injury or visible marks such as bruises	13	5—35
	Fabricated or induced illness by:		
	• misreporting of child's symptoms		
	• interfering with investigations, specimens and treatment		
	• direct interference with, or harming the child so as to produce symptoms and signs		
Sexual abuse		6	25 girls, 8.7 boys
	Penetrative genital or oral contact		
	Non-penetrative genital or genital-oral contact		5—10 girls, 1—5 boys
	Non-contact sexual exposure and exploitation		13 girls, 3.7 boys
Emotional abuse		27	4—9
	Emotional unavailability		
	Hostility and rejection		
	Developmentally inappropriate interactions		
	Exposure to domestic violence		
	Using the child for the fulfilment of the adult's needs		
	Failing to promote the child's socialization		
More than one type		8	

Table 19.2 Differences between various forms of child abuse and neglect.

	Sexual abuse	Physical abuse	Emotional abuse and neglect
Maltreating act or interaction	Hidden	Hidden or observed	Observable
Identity of the maltreating person	Usually in question	Sometimes known	Known
Abuser and primary carer	Usually different persons	Same or different persons	Same person
Immediate protection indicated	Yes	Usually, especially with young children	Rarely

have suffered or witnessed physical violence and to have experienced emotional abuse or disruption to their care [5]. Emotional abuse, physical abuse and neglect are often found in families where one or both parents are suffering from mental illness, have a personality disorder or abuse drugs/alcohol. Violence between parents is also a risk factor [6]. However, no single adult psychopathology is consistently associated with child maltreatment. Sexual abusers are mostly male.

Children of all ages may experience abuse and neglect. Physical neglect and emotional abuse often start early in the child's life, and continue as enduring patterns of care and interaction during childhood and adolescence. Physical abuse in infancy may result from the parent's inability to cope with the demands of the baby; this sometimes causes serious injury and even death. Later in childhood, physical abuse is more associated with inappropriate and harsh punishment. Sexual abuse occurs more commonly in adolescence and in girls, although young boys and girls are also sexually abused.

Fabricated or induced illness (previously known as Munchausen syndrome by proxy, or factitious disorder by proxy) is nearly always perpetrated by mothers, and the child may also have a genuine illness.

Abuse and neglect may be self-limiting or single events but often continue over many years either as a pattern of interaction within a particular parent–child relationship, as a pattern of child-rearing or, in child sexual abuse, as an addiction-like propensity that the same abuser extends towards more than one child.

THE HARM TO THE CHILD

Harm may be caused by a number of mechanisms (Table 19.3). Effects depend on the *child's* genetic vulnerability [9], age and gender; the nature and duration of the *maltreatment*; the *child's*

Table 19.3 Mechanism of harm associated with maltreatment.

Mechanism	Examples
Specific direct effects	Physical abuse causing injury or death
	Emotional neglect leading to emotional withdrawal or indiscriminate affection-seeking
	Effects on the developing brain [7]
	Sexual abuse causing sexually transmitted diseases or unwanted pregnancy
Indirect effects	Effects on later health including obesity, ischaemic heart disease, cancer [8]
	Fabricated or induced illness leading to experience unnecessary investigations and treatments
Effects of the meaning of maltreatment	Sexual abuse leading to a sense of shame, depression, deliberate self-harm
	Hostility and rejection leading to low self-esteem
Effects of associated emotional abuse	Most physical abuse and neglect are accompanied by emotional abuse
Effects of associated carer—child relationship	Sexual abuse may be associated with blame and non-belief of the child by the child's caregivers
Effects of associated social adversity	Poverty, social isolation, migration, natural disasters
Effects of intervention	Effects of removal of the child to inadequate alternative care

relationship to the maltreating person; and the presence of other, *protective* relationships and a supportive social context.

Mental health

The greatest morbidity associated with child maltreatment is psychological, emotional and behavioural [10]. Many maltreated children develop disorganized patterns of attachment [11], which are associated with maladaptive interpersonal relationships. Physical abuse is associated with aggressive behaviour [12] and low self-esteem. Emotional neglect leads to educational underachievement and difficulties in peer relationships as well as to oppositional behaviour. Sexual abuse is particularly associated with later depression, substance abuse and self-harm, post-traumatic phenomena and inappropriate sexual behaviour, with the latter being particularly troublesome in young children [13].

Educational progress and employment

Children and adolescents who have been maltreated, especially by neglect or physical and emotional abuse, underachieve educationally to a significant extent with later poor prospects for optimal employment, even when socioeconomic effects are controlled for [14].

Antisocial behaviour

Antisocial behaviour has been shown to be significantly associated with prior child maltreatment [15].

RECOGNIZING MALTREATMENT

As illustrated in Table 19.2, some forms of maltreatment are readily visible such as neglect and emotional abuse. However, the hallmark of sexual abuse is its secrecy; physical abuse is sometimes observed as it occurs, but is usually recognized by the marks it leaves (Table 19.4).

Parents and abusers do not as a rule report their maltreating actions. When a child presents with difficulties suggestive of abuse or neglect, the process of identification and investigation is usually marked by:

- absence of an acceptable explanation;
- some degree of denial of the possibility of abuse;
- a lack of, or only partial assumption of, responsibility for the child's difficulties by the carer or abuser.

Such responses by the parents or alleged abusers constitute the basic context for the investigation, recognition and management of child abuse and neglect. Many professionals find it difficult to contemplate or accept the possibility that a parent (who may also be a patient) has harmed their own child [16]. Almost invariably, therefore, there is some degree of dispute or doubt during the process of recognizing child maltreatment. This is important not so much for the apportioning of blame, but rather for the subsequent processes of both protecting the child and bringing about change in the relationship between the child and their abuser, if they are to remain living together or in contact. The fact that child maltreatment may also lead to criminal prosecution further complicates matters.

The onus of proof that abuse has occurred often falls on paediatricians, child psychiatrists and psychologists and social workers, sometimes clouding the issue of the child's well-being in favour of parental interests. This is compounded by a societal approach that often favours a narrow child protection approach, which seeks evidence

Table 19.4 How child maltreatment comes to light.

	Neglect	Physical abuse	Emotional abuse	Sexual abuse
Ill-treatment of the child observed	√	May or may not	√	
Harmful effects to the child observed	√	√	May or may not	May or may not
Ill-treatment reported by the child		May or may not		√

of maltreatment, over a family welfare approach. The latter, however, runs the risk of leaving children unprotected. With respect to the recognition of child maltreatment:

- In physical abuse (including faltering growth and fabricated or induced illness) the identification is usually made by paediatricians, radiologists or sometimes retrospectively by pathologists.
- Physical neglect is recognized by the absence of social norms of basic child care and provision.
- Recognition of sexual abuse relies most strongly on the child's verbal descriptions; 80% or more of cases have no conclusive physical signs of abuse. It is therefore the child's words and credibility that are closely tested and challenged. Professionals may receive unexpected disclosures of abuse, usually from children. The appropriate response is to listen but not probe, not to promise confidentiality, but to explain that this information will need to be passed on to social services and to explore misgivings that the child may have about this. It is vital that a written record is made of all such conversations.
- The ill-treatment and harmful interactions in emotional abuse are observable, but it is the extent of their harmfulness that is disputed. Emotional abuse cannot be reliably recognized by the effects on the child, since these are not specific to this form of maltreatment.

Regarding the suspicion of child maltreatment, the National Institute for Health and Clinical Excellence (NICE) divides alerting features into two categories: *Consider* and *Suspect*, and offers good practice guidelines appropriate to each of these categories to both clinicians and other professionals working with children [17].

INTERVENTION

Table 19.5 outlines four tiers of concern with respect to child, family and environmental factors. In order to intervene appropriately, it is helpful to separate the information arousing concern about the child and family into these tiers.

Aims of intervention

The aims of intervention are, if necessary, immediate treatment and immediate protection. They usually also include healing the effects of the maltreatment and protection from future harm.

Immediate treatment

A minority of children who have been maltreated will require immediate medical or psychiatric treatment, including children who have been seriously

Table 19.5 Tiers of concern.

Tier 0
Family and environmental factors:
Including poverty, social isolation, displacement
Tier 1
Parental risk factors:
Including mental ill-health, substance abuse, history of significant own maltreatment, domestic violence [18]
Tier 2
Parent–child interactions:
Forms of maltreatment – emotional abuse, neglect, physical abuse, sexual abuse
Tier 3
Child's functioning:
Aspects that are attributable to maltreatment

injured or infected with a sexually transmitted disease, or who are acutely traumatized by the abuse.

Child protection

As shown in Table 19.2, some forms of maltreatment require immediate protection of the child. The determination of this need is a multiagency endeavour, led by children's social care services. The approach with neglect and emotional abuse is usually to work towards child protection, rather than to gain immediate protection. Protection can be achieved by one of the following:

- A change in the maltreating parent or their circumstances through therapeutic or other work, during which the child will continue to be at risk.
- Supervision of all contact between the child and the abuser, in practice only sustainable for brief periods.
- Separation of the child from the maltreating person, which is therefore the only way of ensuring the immediate safety of the child. However, if the abuser is also the child's primary caregiver, there is a significant cost to achieving immediate protection. Even when the person(s) caring for the child are not the maltreating ones, it is nevertheless necessary to assess their capacity to protect the child from maltreatment by others. The most important determining factor here is the nature of the relationship between the non-abusing caregiver(s) and the abuser. The closer this is the more precarious will the child's position be. 'Closeness' here includes love, but may also mean fear or dependency.

Ensuring protection may, therefore, require statutory measures either by a children's social care protection plan, or through family (civil) court proceedings. The criminal law has little if any part to play in child protection.

Treatment for the effects of maltreatment and prevention of further maltreatment

A comprehensive treatment plan includes:

- Help for the symptomatic child, following protection or accompanying work with the maltreating caregiver to prevent continuation of the maltreatment.
- Work with the maltreating parent(s).
- Support for the non-abusing caregiver(s).
- Work with the whole family, including siblings who may not be (or appear not to be) immediately involved.
- Attention to social/environmental disadvantage.

Evidence for the effectiveness of interventions is variable [19].

As described above, there is no unitary post-abuse syndrome, even following specific forms of abuse such as sexual abuse. Evidence-based therapeutic approaches for the various child and adolescent mental health difficulties are indicated. In particular, children who are experiencing post-traumatic stress disorder (PTSD) and inappropriate sexualized behaviour benefit from trauma-focused cognitive behavioural therapy [20]. The developmental and emotional deficits following neglect need to be addressed, as far as is possible. This may be achievable by supporting the child's parents, providing they are willing to accept help, and may include the treatment of the adults' mental ill health and substance abuse. Experience shows that such help often needs to be maintained for long periods, and that change is not sustained following a short, albeit intensive, course of intervention. Many children also require educational remediation for the associated educational underachievement. Special attention is needed for the depression, substance abuse and self-harm that may develop in adolescence following the experience of childhood or adolescent abuse or neglect.

As well as emotional and behavioural difficulties, many maltreated children also undergo social disruption as a consequence of the necessary protection process. These children are preoccupied with separations and impermanence, and should be involved in age-appropriate decision-making. They require active support through this process.

The child's parents may initially oppose professional intervention. Acknowledging responsibility for the maltreatment, and sometimes for their inability to protect the child, is a difficult and painful process for the parents. They require support and specific therapy geared towards change.

CONCLUSION

Child maltreatment carries a heavy burden of harm to the child, which may continue into adulthood and is a public health issue. Early recognition

and intervention are necessary for prevention or the worst harm. However, recognition and effective management involve a complex process that requires alertness to its possibility and a coordinated, multidisciplinary and multiagency approach. While the family is of central importance to the child's well-being, the child's own interests are paramount and sometimes these may not be achievable within the original family.

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The Neuroscience and Genetics of Childhood Maltreatment

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THE IMPACT OF MALTREATMENT ON BRAIN DEVELOPMENT

A growing body of research has investigated how stress, and specifically different forms of childhood maltreatment, can influence neural structure and function. These studies have employed both children who have experienced maltreatment or adults reporting childhood histories of early adversity. The main brain imaging modalities are summarized in Figure 20.1. In this chapter we focus on studies of children, first considering those that have investigated differences in brain structure, followed by the smaller number of studies that have investigated the potential impact of maltreatment on brain function.

STRUCTURAL DIFFERENCES

Subcortical structures: hippocampus and amygdala

Animal research has shown that the hippocampus plays a central role in learning and various aspects of memory and that these functions are impaired when animals are exposed to chronic stress. Studies of adults with post-traumatic stress disorder (PTSD) who have histories of childhood maltreatment, an early form of stress, consistently report that these individuals have smaller hippocampal volumes. It is surprising then that structural magnetic resonance imaging (sMRI) studies of children and adolescents with maltreatment-related PTSD consistently fail to detect decreased hippocampal volume [1]. It is possible that the impact of stress is delayed and becomes manifest only later in development.

The amygdala, another key subcortical structure, plays a central role in evaluating potentially threatening information, fear conditioning, emotional processing and memory. Given that experiences of maltreatment typically occur in family environments characterized by unpredictability and threat, it might be expected that children growing up in such contexts would show increased amygdala volume, comparable to that found in stress-exposed animals, which show increased dendritic arborization [2]. However, a recent meta-analysis of children with maltreatment-related PTSD did not find significant differences in amygdala volume between maltreated and non-maltreated children [3]. By contrast, more recent studies *have* reported an increase in amygdala volume in maltreated children (see, e.g., Ref. [4]), suggesting that perhaps such effects are subtle and difficult to detect reliably or are associated with heightened forms of adversity experienced as a result of institutionalization.

Cortical structures: prefrontal cortex and cerebellum

The prefrontal cortex (PFC) plays a major role in the control of many aspects of behaviour, regulating cognitive and emotional processes

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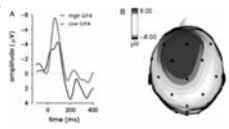
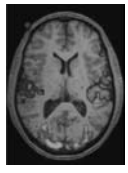
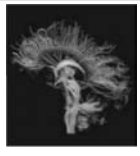
Imaging modality	How it works	Advantages	Disadvantages
ERP 	Records the brain's electrical activity and yields detailed information about the temporal sequence (resolution in milliseconds) of cognitive operations throughout the brain	• Non-invasive • High temporal resolution • Low cost • Easy to use with children to study early brain functioning	• Poor spatial resolution • Limited to surface (cortical) activity
fMRI 	Detects the changes in blood oxygenation and flow that occur in responses to neural activity	• Non-invasive and no radiation • High spatial resolution	• Poor temporal resolution • Analyses are complex and time consuming • Very susceptible to movement • Many contraindications • High cost • Indirect measure of brain functioning
DTI 	Provides information about the structural integrity of axonal white matter by measuring the molecular diffusion of water in brain tissue	• Non-invasive • High spatial resolution	• Difficult to conduct group comparisons • High cost • Only an indirect measure of the structural integrity of white matter

Figure 20.1 An overview of the characteristics, advantages and disadvantages of the main brain imaging modalities used to investigate the impact of childhood maltreatment. ERP, event-related potential; fMRI, functional magnetic resonance imaging; DTI, diffusion tensor imaging.

through extensive interconnections with other cortical and subcortical regions.

There are mixed findings from studies comparing the PFC volume of children with maltreatment-related PTSD and non-maltreated children. Recent studies have reported smaller prefrontal volume associated with the experience of maltreatment (e.g. Ref. [5]), and less prefrontal white matter, while other studies have reported larger grey matter volume of the middle-inferior and ventral regions of the PFC in maltreated groups. There are several possible reasons for these inconsistent findings and it is likely that methodological differences across studies, including the use of different imaging techniques and age groups of children, might at least partly account for these reported differences [6]. It is also possible that there are regionally specific windows of vulnerability in brain development; the frontal cortex undergoes significant structural change during adolescence. We know that sexual abuse, for example, during the adolescent period is more associated with structural differences in this brain region compared to other regions [7]. Unfortunately, most brain imaging studies have not systematically considered the age at which different kinds of abuse have occurred; from a clinical perspective it would be helpful for further research to systematically investigate the relative vulnerability of different brain regions at different ages to different forms of early adversity.

By contrast, decreased volume of the cerebellum in children and adolescents with a history of maltreatment has been a consistent finding in the literature [6], chiming with growing evidence that this structure plays a crucial role in emotion processing and fear conditioning via its connection with limbic structures and the hypothalamic–pituitary axis (HPA). The cerebellum has also been shown to be involved in executive functioning, which is impaired in children with a history of maltreatment [8].

Corpus callosum and other white matter tracts

The corpus callosum (CC) is the largest white matter structure in the brain and controls inter-hemispheric communication of a host of processes, including, but not limited to, arousal, emotion and higher cognitive abilities. With the exception of one study, decreases in CC volume have consistently been reported in maltreated children and

adolescents compared to non-maltreated peers [1]. Recent studies that have employed diffusion tensor imaging (DTI) have found differences in maltreated children in frontal and temporal white matter regions, including the uncinate fasciculus, which connects the orbitofrontal cortex to the anterior temporal lobe, including the amygdala (e.g. Ref. [9]). The extent of the white matter differences observed by Govindan and colleagues was associated with longer periods within an orphanage and may underlie some of the socio-emotional and cognitive impairments exhibited by maltreated children.

FUNCTIONAL DIFFERENCES

In contrast to the number of studies examining structural brain differences, only a few have investigated possible functional correlates associated with maltreatment using brain imaging techniques such as functional MRI (fMRI) or electrophysiological techniques.

fMRI studies

To date, five fMRI studies have compared maltreated children to non-maltreated children. Building on the experimental evidence that maltreated children show hypervigilance to threatening facial cues, two fMRI studies have examined the neural correlates of face processing in this population. These studies have reported that maltreated children are characterized by increased amygdala response to threatening cues in comparison to non-maltreated children [10] – findings consistent with amygdala volume differences observed in the structural MRI studies reviewed above. Two other studies assessed response inhibition and observed increased activation in the anterior cingulate cortex (ACC) in maltreated youths as compared to controls. These results suggest impaired cognitive control in maltreated youths, which, in turn, could confer risk for psychopathology [11], especially in the context of heightened subcortical responses such as that observed during affective processing. The fifth study used a verbal declarative memory task and compared youths with post-traumatic stress symptoms (PTSS) secondary to maltreatment with healthy controls [12]. During the retrieval component of the task, the youths with PTSS exhibited reduced right hippocampal activity, which was associated with greater severity of avoidance and numbing symptoms.

Event-related potential (ERP) studies

Much of the existing ERP research has compared the pattern of brain response of adversely treated children and healthy children when processing facial expressions, an ability that is usually mastered by the preschool years. When compared with non-institutionalized peers, institutionalized children who have experienced severe social deprivation showed a pattern of cortical hypoactivation when viewing emotional facial expressions, and familiar and unfamiliar faces [13]. In contrast, a second set of important studies has provided convincing evidence that school-aged children who had been exposed to physical abuse show increases in brain activity specific to angry faces and require more attentional resources to disengage from such stimuli (e.g. Ref. [14]). These ERP findings are consistent with recent fMRI evidence and suggest

Box 20.1 Summary of structural and functional brain differences associated with maltreatment

In summary, there is relatively consistent evidence for reduced white matter and reduced grey matter volume in the cerebellum of maltreated children, but no differences in relation to the hippocampus. The structural findings are more mixed for the PFC. Functional brain imaging research suggests that experience of maltreatment is associated with increased amygdala and anterior cingulate cortex response in affective and cognitive control paradigms, respectively. Event-related potential (ERP) studies have found that children who have experienced severe social deprivation show a generalized pattern of cortical hypoactivation. Increased brain activity, specifically to angry faces in prefrontal regions, has also been observed in physically maltreated children, likely to represent the neural correlates of increased attentional monitoring for social threat.

that some maltreated children are allocating more resources and remain hypervigilant to potential social threat in their environment, likely to be at the cost of other developmental processes.

THE ROLE OF GENETIC INFLUENCES

It is a common but often striking clinical experience to find that two children who have experienced very similar patterns of early adversity have very different outcomes. While this may be partly due to specific environmental or psychological factors characterizing one child, but not the other, there is increasing evidence that such differential outcome may in part at least be due to genetic differences.

We now know that many of the psychiatric outcomes that are associated with maltreatment, such as PTSD, depression and antisocial behaviour, are partly heritable. However, it is incorrect to think that there are particular genes for these disorders. Rather, we are learning that there are a wide number of genetic variants that may subtly alter the structure and functioning of neural circuitry and hormonal systems that are crucial in calibrating our individual response to social affective cues, and in regulating our stress response [15]. In recent years, researchers have focused in particular on the way in which such genetic variants and adverse environments may interact. Such gene by environment (G × E) interaction research has demonstrated that for a range of genetic variants (known as polymorphisms) childhood maltreatment can increase the risk of later psychopathology for some children more than others. For example Caspi and colleagues [16] were the first to report on an interaction of a measured genotype (*MAOA*, monoamine oxidase A) and environment (maltreatment) for a psychiatric outcome and demonstrated that individuals who are carriers for the low-activity allele (*MAOA-l*) were at an increased risk for antisocial behaviour disorders following maltreatment. Imaging genetic studies have found that the risk genotype, *MAOA-l*, is related to hyper-responsivity of the brain's threat detection system and reduced activation in emotion regulation circuits. This work suggests a neural mechanism by which the *MAOA* genotype engenders vulnerability to reactive aggression following maltreatment [17].

In other words, G × E research suggests that a child's genotype may partly determine their level of risk and resilience for adult psychiatric outcomes, including depression and PTSD following childhood maltreatment (e.g. Ref. [18]). It is important to bear in mind, however, that positive environmental influences, such as social support, can promote resilience, even in those children carrying 'risk' polymorphisms exposed to maltreatment [18]. This finding illustrates the important point that when considering a G × E interaction, positive environmental influences (such as contact with a supportive attachment figure), are as relevant to consider as negative environmental influences such as maltreatment. Future research will investigate the influence of clinical interventions as a positive environmental factor that may serve to moderate environmental and genetic risk.

CLINICAL IMPLICATIONS

There is accumulating evidence pointing to a variety of neurobiological changes associated with childhood maltreatment. Such changes can, on the one hand, be viewed as a cascade of deleterious effects that are harmful for the child; however, a more evolutionary and developmentally informed view would suggest that such changes are in fact adaptive responses to an early environment characterized by threat. If a child is to respond optimally to the challenges posed by his/her surroundings then early stress-induced changes in neurobiological systems can be seen as 'programming' or calibrating those systems to match the demands of a hostile environment. From a clinical perspective, such adaptation may heighten vulnerability to psychopathology, partly due to the changes in how emotional and cognitive systems mediate social interaction [19]. For example, early-established patterns of hypervigilance, while adaptive in an unpredictable home environment, may be maladaptive in other settings, thus increasing vulnerability for behavioural, emotional and social difficulties.

While initial research has focused on these neurobiological adaptations following maltreatment, there is increasing interest in exploring the concept of resilience and those factors that may promote or enhance neurobiological mechanisms important for emotional regulation and coping. Specifically, there is emerging evidence from genetic and neurobiological research supporting the importance of a reliable adult caregiver, and the role they

can play in helping to scaffold the child's ability to regulate stress [18,20]. Such work will begin to shed light on how professionals can intervene more effectively to promote better systemic structures around children that improve resilience and moderate the impact of maltreatment. We are likely to see an increasingly fruitful dialogue between developmental research, focused on a child's psychological representations of their social world, and neurobiological research, focused on putative neural mechanisms underlying adaptive responses to stress and effective emotional regulation.

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Section 5

Atypical Development

Section 5a

Infancy and Early Childhood

21

Feeding and Eating Disorders in Infancy and Childhood

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INTRODUCTION

Feeding behaviour develops and is best understood within a bio-psycho-social context. Normal feeding requires the successful integration of healthy, developing physical and psychological function, as well as a facilitative interpersonal context. Normal childhood eating is not well defined, and there are limited data to help determine the edges of disorder, or 'caseness'. Additionally, some parents or caregivers might experience their child's eating behaviours as problematic, yet on clinical assessment the child might be deemed to be presenting with feeding or eating behaviours well within the normal range. In such instances, interactions around feeding can become dysfunctional, requiring clinical intervention. It has been suggested that early feeding and eating disturbances might helpfully be approached and understood more explicitly within an interpersonal context rather than being located solely in the child [1]. The distinction between transient feeding problems and those likely to become more chronic and severe is often difficult to make; there is almost no research evidence underpinning the identification of presenting features, or combinations of features, associated with a poorer prognosis or response to treatment.

PRESENTATION

In clinical practice children present with a range of feeding and eating difficulties (see Box 21.1), many of which are of uncertain nosological status [2].

Delayed or absent feeding skills can result from different factors. Some children with developmental disorders or specific medical conditions might be unable to drink or eat due to delay or dysfunction in their ability to latch, suck, chew or swallow. Others might present with delay in feeding skills due to having experienced enteral feeding. Not uncommonly children remain dependent on tube feeding longer than is medically necessary due to missed learning opportunities or lack of a timely structured programme of tube weaning. Children who remain tube dependent beyond the age of 5 years experience particular difficulties with tube weaning [3]. A few children present with delayed feeding skills because they have not been offered opportunities to progress with feeding development.

A number of children present with difficulty in managing or tolerating ingested fluids or foods, as evidenced by gagging, retching, choking or vomiting. Some children brought to clinical attention for a feeding disorder have a previously undiagnosed underlying intolerance or other gut problem, resulting in diarrhoea, constipation or abdominal discomfort. Reluctance to feed can significantly resolve with appropriate medical management of physical symptoms where indicated. Where no physical causes can be identified, psychological or behavioural treatment approaches are more appropriate.

Lack of appetite or disinterest in food can also be associated with a number of different factors encompassing other mental and behavioural

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Box 21.1 Common childhood feeding and eating disorder presentations

- Delayed or absent feeding skills
- Difficulty managing or tolerating ingested fluids or foods
- Lack of appetite or interest in food
- Avoidance or refusal to eat based on sensory aspects of food
- Fear-based avoidance or refusal to eat based on aversive consequences
- Utilizing specific behaviours related to feeding/eating to comfort, self-soothe or self-stimulate.

disorders such as pervasive developmental disorder, hyperkinetic disorder or attachment disorder, as well as chronic medical conditions and use of certain medications. In infants, lack of appetite or interest in food might be associated with regulatory difficulties, such as excessive crying or sleeping. Infants experiencing parental neglect, stress or trauma may fail to develop healthy hunger-satiety responses, presenting with feeding problems and associated growth delay. However, some children present with a clear lack of interest in food and eating but are otherwise developing and functioning normally. Such children fail or refuse to eat sufficient amounts, tend not to communicate hunger, and can present with faltering growth and in some cases extreme underweight. This presentation is seen throughout childhood, and in older children is often associated with sadness, worry and other emotional difficulties.

Avoidance or refusal to eat based on sensory aspects of food is a relatively common presentation, and is referred to by a number of different terms in the literature, such as sensory food aversion, or selective eating. Children with this type of presentation consistently refuse foods based on texture, taste, appearance, smell or temperature. In general such children have a relatively restricted range of preferred foods, which they will eat without difficulty. Often weight and growth are normal, but the accepted diet can be deficient in essential vitamins or minerals and/or be excessively high in fats, salt or sugar. This type of presentation is relatively common in children with autism spectrum disorders, but also occurs in children without such a diagnosis. At times of pressure or stress, the accepted range may be reduced to a smaller number of 'safe' foods.

Children who have had traumatic or unpleasant experiences involving the gastrointestinal tract (e.g. frequent vomiting, nasogastric tube feeding, suctioning) may present with an extreme unwillingness or reluctance to eat. On observation such children may display typical fear-based avoidance behaviours, or safety behaviours such as very slow chewing or reluctance to accept anything other than smooth textures. Food refusal or avoidance is related to past experience, and associated expectation or concern about aversive consequences of eating. In some cases these presentations can be conceptualized as a specific phobia, and can be helpfully formulated and treated as such.

Finally, a number of children present with behaviours related to feeding or eating where the primary function seems to be to derive some level of comfort, self-soothing or self-stimulation. Such behaviours include the eating of non-nutritive substances, as in pica (see 'Diagnosis' below). This is often found in association with mental retardation, but not exclusively so, as it is also seen in children of normal cognitive ability. Repeated regurgitation and re-chewing, and re-swallowing of food, as in rumination disorder (see 'Diagnosis' below) appears to serve a similar function.

DIAGNOSIS

Children with feeding difficulties tend to present in a range of different clinical settings and are seen by clinicians from a range of specialties, which has contributed to a wide array of descriptive and diagnostic terms being used often for very similar presentations. 'Feeding Disorder' is the diagnostic term used in ICD-10 (*International Classification of Mental and Behavioural Disorders in Children and Adolescents*: F98.2 Feeding disorder of infancy and childhood) [4] and DSM-IV-TR (*Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition – Text Revision*: 307.59 Feeding Disorder of infancy or early childhood) [5]. Both sets of criteria require failure to eat adequately associated with weight loss or failure to gain weight over a period of at least 1 month prior to presentation. Both state that the failure to eat adequately is

not directly due to another medical condition or another mental disorder, and both require onset of the disturbance before the age of 6 years. In practice, many children presenting with significant feeding problems do not present with low weight, and they therefore fail to meet diagnostic criteria. The mismatch between what is seen in clinical practice and current diagnostic criteria is illustrated by a study demonstrating that only around 12% of children presenting to one feeding disorders clinic met DSM-IV-TR criteria for Feeding Disorder [6].

Other disorders currently included under the general heading of feeding disorders include Pica and Rumination Disorder. Pica is included in ICD-10 in the mental and behavioural disorders section; it is also included in DSM-IV-TR. Rumination Disorder is included in DSM-IV-TR, but does not appear as a separate diagnosis in the mental and behavioural disorders section of ICD-10. The diagnostic criteria for ICD-10 Feeding Disorder include mention of rumination, and the section Feeding Problems of Newborn (P92) includes Regurgitation and Rumination in Newborns, although not identified as a behavioural or mental disorder. Both pica and rumination presentations are seen in young children, but are also seen in older individuals. Current proposals for DSM 5 include revising the placement of these two disorders so that they are no longer identified as disorders of infancy or early childhood alone [7].

Some clinicians working with young children with feeding disorders prefer to use the Zero to Three system (DC:0–3R) [8]. This is a diagnostic classification system specifically for mental health and developmental disorders of infancy and early childhood. It includes a section on Feeding Behaviour Disorder and provides descriptive criteria for six subcategories (Feeding Disorder of State Regulation; Feeding Disorder of Caregiver-Infant Reciprocity; Infantile Anorexia; Sensory Food Aversions; Feeding Disorder Associated with Concurrent Medical Conditions; Feeding Disorder Associated with Insults to the Gastrointestinal Tract). Finally, a number of children with feeding disorders present to paediatric services, in particular paediatric gastroenterology clinics. Rome III is an internationally established system for the diagnosis and classification of functional gastrointestinal disorders (FGIDs) [9], which overlaps with behavioural and mental disorder classifications. Relevant to this chapter is the overlap between Infant Rumination Syndrome (in Rome III as a FGID), Regurgitation and Rumination in Newborns (ICD-10) and Rumination Disorder as a mental/behavioural disorder in DSM-IV-TR.

ASSESSMENT

Due to the complex nature of feeding, clinical assessment must include information across a number of areas. Box 21.2 summarizes core recommended components of assessment for a childhood feeding or eating disorder.

There are few standardized, well-validated assessment measures relating to early feeding and eating difficulties. There are no widely used diagnostic interviews, and the majority of instruments are parent-completed questionnaires such as the Behavioral Pediatrics Feeding Assessment Scale [10]. Observation of a feeding situation is extremely useful.

EVALUATION OF RISK AND PRIORITIZATION OF AREAS FOR INTERVENTION

Box 21.3 includes the main domains of development and function usefully considered by clinicians in relation to determining clinical risk and prioritization of areas for intervention.

Determining the nutritional adequacy of a child's diet includes an estimation of the overall energy intake as well as a nutritional breakdown. Specific nutritional deficits, for example as in iron deficiency anaemia, should be treated. It is important to note that children's energy needs vary considerably between individuals. The main issue for the clinician is to determine whether the child is at short- and/or longer-term physical risk if current intake continues, and if so to put in place steps to improve nutritional adequacy. This might be through supplementation, behavioural or psychological interventions, or a combination of these.

Childhood feeding and eating difficulties can have varying effects on weight, growth and physical development/function; if growth is clearly dropping down centiles, intervention is indicated. Other aspects of development are also important to consider; for example, is a dependence on soft or

Box 21.2 Core components of clinical assessment interview

- Current feeding – oral, tube-fed, self feeding, etc.
- Energy and nutritional breakdown of current intake
- Weight, height, BMI (body mass index) centile
- Developmental history – including early feeding and any history of forced feeding
- Medical history – including oro-motor/swallow problems, history of reflux, vomiting, tube feeding, allergies/intolerances
- General behaviour – at home and at nursery/school, including child's attitude to messy play, signs of oral aversion (e.g. refusal to brush teeth)
- Caregiver concept of problem (e.g. medical/psychological), expectations and previously tried strategies
- Relevant family medical history – atopy, intolerances, feeding problems in siblings
- Structure of family mealtime and family attitudes to mealtimes
- Caregiver mental health and food/eating/weight history
- Family life events and caregiver support network
- Observation of mealtime situation to assess feeding interaction between child and caregiver
- School/nursery/relevant other views about child and ability to offer support around feeding.

pureed foods holding the child back in terms of developing appropriate biting and chewing skills?

Some children with eating difficulties might not present with nutritionally inadequate diets or weight or growth impairment, for example where there is a continued dependence on toddler or baby foods in a school-aged child. In such cases the eating problem can have a significant negative impact on social and emotional development and function. The child might refuse to mix with peers at mealtimes and miss out on important social and educational occasions, such as visiting friends or going on school outings. Such events are important in relation to developing independence, autonomy and social competence. Some children present with increasing anxiety or distress in relation to their eating problems, experiencing embarrassment, low mood or frustration. Irrespective of the presence or absence of physical sequelae of the eating difficulty, such features form an important focus for clinical attention.

Box 21.3 Main domains for consideration at presentation

Recommended areas for consideration at assessment in relation to risk and prioritization of intervention:

- Nutritional adequacy of intake (overall energy intake and nutritional breakdown).
- Impact of feeding/eating disturbance on weight, growth and physical development/function.
- Impact of feeding/eating disturbance on social and emotional development/function.
- Impact of feeding/eating disturbance on interaction with caregiver and family function.

It is well recognized that parenting a child with feeding or eating difficulties can be stressful and distressing. In some families, interactions between child and caregiver can become fraught and difficult, and parents may feel anxious, frustrated or inadequate. Family social behaviour around

food and eating can be affected, and disagreements between caregivers about management of eating behaviour can contribute to family tension and conflict. This can carry with it a risk of the child being hit, risk of precipitating or exacerbating parental mental health problems, increased risk of relationship difficulties between parents and other close family members, and the emergence of behaviour problems in siblings.

TREATMENT

There is a very poorly developed evidence base for feeding disorder treatment interventions. A review of the literature on treatment of childhood feeding disorders characterized by significant refusal or selectivity highlights that the majority of publications over the past few decades have been based on single case research [11]. There are no well-designed, well-controlled treatment studies of large cohorts of well-defined cases. In general there is a lack of standardized, replicable interventions. Behavioural interventions are common, but other areas are important in intervention, as summarized in Box 21.4 [12]. In general, comprehensive, multifaceted approaches are required, which are usually delivered in a multidisciplinary team context [13].

CONCLUDING COMMENTS

It is often difficult for clinicians to clearly separate out somatic and psychological aspects of presentations, and a significant number of children presenting with clinically significant difficulties fail to meet diagnostic criteria. The prevailing situation regarding classification and terminology represents a significant problem for the field and has contributed to a relatively poor state of knowledge with regard to treatment interventions, course, prognosis and outcome. This represents a major challenge and a priority for further work. In addition robust, well-validated assessment tools are lacking, making it difficult to obtain reliable incidence and prevalence rates. There are few long-term follow-up studies of early feeding problems from which to gauge longer term risks, and very limited longitudinal data of specific types of feeding difficulty leading to relatively poor knowledge about course and prognosis. We do know, however, that behavioural interventions are often associated with significant improvements in feeding behaviour [11], and with proposals for DSM 5 and ICD-11 on the horizon, new opportunities for research in relation to treatment and outcome may be stimulated.

Box 21.4 Core components of treatment

- Improve nutritional status
- Facilitate development of appetite/skills acquisition opportunities
- Behavioural/psychological interventions
- Educate and support caregivers
- Liaison with wider system, to include school, other care providers

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Literacy Disorders

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WHAT'S NEW

- Literacy disorders are now recognized as dimensional, with no clear cut-off between typical and impaired reading; 'diagnosis' is defined according to context, e.g. the school system.
- Disorders of reading accuracy stemming from phonological (speech sound processing) difficulties are partially independent from disorders of reading comprehension, which are associated with broader oral language impairments.
- Literacy disorders tend to co-occur alongside other learning difficulties (e.g. maths disorders and attention disorders).
- Different forms of literacy disorder require different interventions; there is now an evidence base of effective interventions to promote phonological skills and for treatments that foster language (especially vocabulary) development.
- Protective factors play an important part in improving the outcome for children with literacy disorders. These include early identification and high-quality intervention, the child's ability to maintain attention and motivation, conditions that foster high levels of print exposure, encouragement to engage in activities in which the child might excel, and family support.
- Children who have severe and complex literacy problems (treatment non-responders) will need intensive and high-quality learning support that is sustained into the teenage years and beyond.

DEFINITION, INCIDENCE, PERSISTENCE AND CO-OCCURRENCE

Disorders of literacy are arguably the most studied and best understood of all the cognitive disorders of childhood. In this chapter we shall focus on both disorders of reading accuracy (dyslexia) and reading comprehension difficulties. Dyslexia is a common disorder affecting around 3–6% of children, with an over-representation of boys [1]. A simple definition of dyslexia is a learning disorder that primarily affects reading and spelling development. A more precise definition used by the International Dyslexia Association views dyslexia as 'a specific learning difficulty of neurobiological origin [. . .] characterised by difficulties with accurate and/or fluent word recognition and poor spelling. These difficulties typically result from a deficit of the phonological component of language.' The notion that in dyslexia 'reading achievement is

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substantially below expectation given the person's chronological age, measured intelligence and age-appropriate education' has now fallen from favour and this discrepancy definition may not be used in DSM-5 (*Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* – in preparation). It is now recognized that dyslexia occurs across a wide range of abilities; dyslexia is not an 'all-or-none' category but rather a dimensional disorder with a number of behavioural outcomes. The child's educational history is also of critical importance in determining the clinical picture and it should be borne in mind that inadequate teaching may be a sufficient explanation for some children's poor reading skills.

Dyslexia is a life-long disorder, and many affected individuals experience problems of reading fluency and spelling that persist into adulthood despite intervention. Dyslexia commonly co-occurs alongside other learning difficulties, such as specific language impairment (SLI), mathematical problems, attention deficit hyperactivity disorder (ADHD) or motor difficulties [2].

The goal of reading is not only to access the printed word, but also to extract meaning from it. It is estimated that 7–10% of middle school-age children can read words accurately but fail to understand what they read [3]. These poor comprehenders are often 'hidden' within their classrooms because their fluent reading masks underlying difficulties.

ACQUIRING LITERACY SKILLS

In order to understand why children fail to learn to read accurately and with understanding, it is important to have a clear picture of typical reading development. The Simple View of Reading proposes that variations in reading development can be understood in terms of two relatively separate underlying skills: word recognition and language comprehension.

Two critical foundation skills for acquiring *word recognition* skill during the first 2 years at school are phoneme awareness (the ability to analyse speech sounds within words) and letter-sound knowledge. Together these skills in pre-school years account for almost 90% of the variance in reading skill at age 6 years. Performance on speeded naming tasks (naming pictures, colours, letters or digits as rapidly as possible) is also predictive of individual differences in reading, especially of reading fluency.

While learning to recognize printed words depends largely on creating 'mappings' between orthography and phonology, the development of *reading comprehension* depends on broader oral *language* skills, such as knowledge of word-meanings, the ability to understand sentences, to make inferences where appropriate and to remember what was read in order to create an integrated and cohesive sense of the text [4].

It has been argued that the ease of acquiring fluent and accurate reading depends on the writing system in which the child is learning [5]. English orthography contains inconsistent letter-sound mappings and permits irregular forms; it is therefore classified as 'opaque'. In contrast, languages such as Italian, Greek and German have a 'transparent' writing system in which sound-to-letter correspondences are regular and consistent. Empirical studies suggest that it is harder to learn to read in English than in transparent orthographies but findings are difficult to interpret because of variations in cultural practices surrounding reading instruction in different languages. The child with dyslexia learning in a transparent (as opposed to opaque) orthography would be expected to have fewer difficulties in learning about spelling-to-sound consistencies, but may nonetheless have persisting difficulties with reading fluency.

THE NATURE OF IMPAIRMENT IN CHILDREN WITH LITERACY DISORDERS

Children with dyslexia fail to learn to read because of an underlying weakness in their phonological system. This weakness is indicated by poor performance on a wide range of phonological tasks, such as verbal short-term memory tests, deleting specified phonemes from words, speeded (rapid) naming and repeating nonwords. Difficulties in processing, memorizing and analysing speech segments in words invariably results in problems of learning to decode in children with dyslexia. The most direct means of investigating this decoding deficit is to ask children to read nonwords like '*kig*' and '*ploob*'. Children with dyslexia have great difficulty reading nonwords, even when compared with younger children. Children with dyslexia learning to read in transparent orthographies tend to have fewer difficulties in

word decoding, but are still likely to have problems with reading fluency and spelling [6].

In contrast to children with dyslexia, poor comprehenders perform well on tests of phonological skills. They do, however, experience problems with a wide range of language-related tasks that assess oral language (vocabulary, grammar and oral expression), higher level language skills (including narrative and use of figurative language), metacognitive processes (integration and inference making, knowledge of story conventions and structures) and executive processes (verbal working memory, suppression and inhibition). Nation *et al*. [7] carried out a longitudinal study of poor reading comprehenders from the ages of 5 to 8 years; the children assessed as having reading comprehension difficulties at age 8 showed oral language problems that were present at school entry 3 years earlier. Such findings suggest that language problems are causally related to later reading comprehension difficulties.

Like most developmental disorders, dyslexia and reading comprehension difficulties occur along a continuum of severity. In dyslexia, the severity of the child's phonological deficit will influence the extent of their reading and spelling difficulties, and very likely also their response to remedial intervention. However, other cognitive factors also play a role. Children with dyslexia who have comorbid language impairment will present with problems in reading comprehension as well as word recognition. Pennington and Bishop [8] have suggested it is important to consider the number and type of 'risk' factors present in a child with a reading problem (these could be genetic or environmental). In considering the comorbidity of dyslexia, language impairment and speech sound disorders, they conclude that there are some risk factors that are general to all three disorders, especially difficulties in acquiring phoneme awareness (see Table 22.1). However, there are also risk factors that are specific to particular disorders; a deficit in rapid naming is specific to children with dyslexia, but is not evident in most children with language impairment.

Table 22.1 Risk factors for dyslexia in the preschool and early school years.

Stage of development	Risk factors for dyslexia
Birth	Affected family member
Preschool	Late talker
	Speech difficulties
	Slow to learn colours and letters
School entry	Poor knowledge of letters
	Poor rhyming or phoneme skills
	Expressive language difficulties

PATTERNS OF IMPAIRMENT FROM PRESCHOOL TO ADOLESCENCE

It has been known for many years that dyslexia runs in families, and recent studies suggest there is a 40% risk of dyslexia developing in first-degree relatives [9]. While dyslexia is most usually diagnosed in middle childhood it is clear from prospective longitudinal studies that its effects are evident as early as 3 years of age and persist through adolescence into adulthood.

Although there are different pathways to literacy development, studies of children at familial risk of dyslexia typically show that children who go on to develop reading problems experience delayed language development in the preschool years [10]. These subtle language problems may persist into the school years when they are associated with phonological impairments at the time of reading instruction. In one such study, language-delayed preschoolers experienced persisting literacy difficulties into early adolescence, when they also tended to present with low self-esteem in relation to their academic skills, avoidance of reading, and attentional and emotional difficulties [2]. Importantly, children from 'at-risk' families who were reading within the normal range at the age of 8 years, went on to experience difficulties in spelling and reading fluency at the age of 12, thus showing a 'broader phenotype' of dyslexia. These findings indicate that dyslexia is a dimensional disorder and that the family risk of dyslexia is continuous. Children from 'at-risk' families who were not reading impaired in the early years tended to have relatively good oral language skills. It seems that their good semantic knowledge enabled them to develop compensatory strategies; for instance, they were able to draw on context cues available in text to aid and support reading processes.

ASSESSING LITERACY DISORDERS

Most children with literacy disorders are referred for assessment in the middle school years. However, there is increasing recognition of the importance of early identification before the child has fallen too far behind educationally and begins to experience declining levels of motivation and confidence. Since early screening batteries tend to have low reliability, a recently commissioned review for the UK government [11] recommended the identification of 'at-risk' children via close monitoring of their response to reading instruction during the first 2 years at school. Children who fail to progress sufficiently in response to mainstream, differentiated and additional literacy support require further assessment for likely dyslexia or reading comprehension disorder (Figure 22.1).

A brief diagnostic assessment of dyslexia should include tests of single word reading and spelling, a test of phonological awareness, a test of short-term verbal memory and an (optional) test of arithmetic. In addition, using a short-form IQ test makes it possible to determine whether the child has a general learning difficulty or a more specific developmental disorder [12].

In order to determine whether a child may have a *complex* learning difficulty, a more comprehensive evaluation is needed. Such an assessment needs to recognize both the multiple components of literacy assessment and the dimensional nature of disorders; not all components of literacy skill

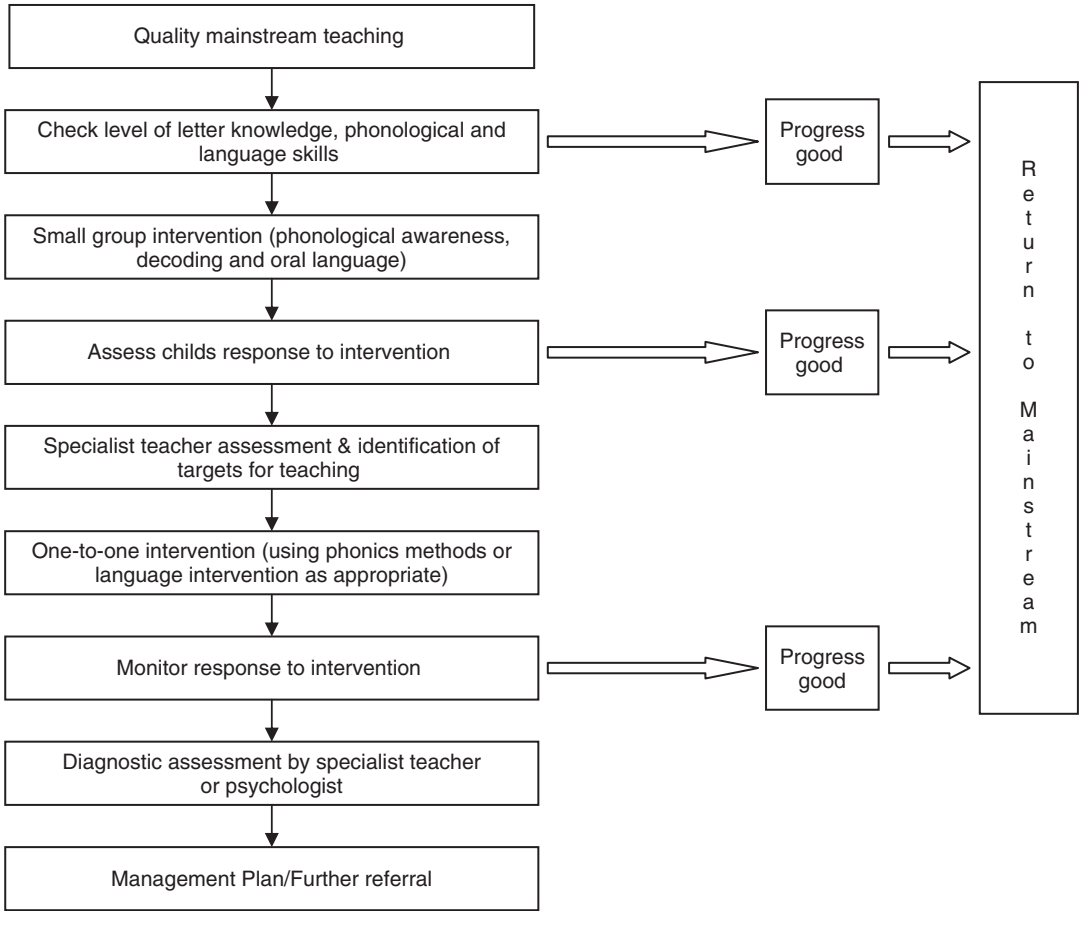
Quality mainstream teaching
Check level of letter knowledge, phonological and language skills
Progress good
Small group intervention (phonological awareness, decoding and oral language)
Assess childs response to intervention
Progress good
Specialist teacher assessment & identification of targets for teaching
One-to-one intervention (using phonics methods or language intervention as appropriate)
Monitor response to intervention
Progress good
Diagnostic assessment by specialist teacher or psychologist
Management Plan/Further referral
Return to Mainstream

Figure 22.1 Steps in the early identification and assessment of literacy disorders: a staged process.

Table 22.2 Components of a comprehensive diagnostic assessment for reading disorders.

Construct	Test
Reading	Single word reading
	Nonword reading (decoding)
	Reading fluency (single words and continuous text)
	Prose reading accuracy
	Reading comprehension (literal and inferential)
Spelling	Single word spelling
	Spelling in free writing
Writing	Written narrative (structure and organization)
	Writing speed
	Quality analysis of handwriting
Mathematics	Mental arithmetic
	Mathematical reasoning
	Written arithmetic
Phonological skills	Phonological awareness
	Rapid naming
Verbal working memory	Verbal short-term memory span
	Nonword repetition
Language skills	Sentence repetition
	Grammar and morphology

will be equally impaired in a given child (see Table 22.2). For the child with suspected dyslexia, the nature of the child's underlying difficulty can be assessed using measures of phonological awareness, short-term verbal memory, rapid naming and decoding (nonword reading). For the child with reading comprehension difficulties, it is important to evaluate their oral language skills and text comprehension strategies. Finally, in view of the common co-occurrence of developmental disorders, the assessment needs to determine which particular additional difficulties the child might have that will impact on their behaviour and school adjustment. Given time constraints, useful information can be sought from parents and teachers using standardized questionnaires that assess, for instance, attention or language difficulties.

TEACHING CHILDREN WITH LITERACY DISORDERS

There is a growing evidence base of effective interventions for literacy disorders. Intervention programmes with proven efficacy for children with dyslexia emphasize the integration of text level reading with training in phonological awareness and decoding skill [13]. Children with reading comprehension difficulties need a broad and rich language curriculum, with a strong emphasis on developing vocabulary knowledge and text comprehension skills. A recent intervention study [14] demonstrated that children's comprehension skills can be significantly improved with a programme that specifically targeted oral language skills (vocabulary enrichment, listening comprehension, use of figurative language and spoken narrative) and text comprehension strategies (including metacognitive strategies such as re-reading and mental imagery, reading comprehension strategies, and making inferences from text). Preschool programmes aimed at strengthening the foundations of literacy (specifically oral language skills and phoneme awareness) can also have positive effects [15].

While teaching programmes are rightly expected to attempt to remediate the child's underlying deficit, there is nonetheless a place for encouraging children to draw on their cognitive strengths so that they can develop compensatory strategies. The verbally able child with dyslexia encountering an unfamiliar word may be taught

a strategy of combining a partial decoding strategy with their vocabulary knowledge and the sentential framework within which the word is embedded. This enables them to make the 'best possible estimate' as to its actual pronunciation.

As children get older, their needs extend beyond direct teaching of reading and spelling to include instruction in the use of information technology (computers, spell-checks and voice-activated software) and techniques for improving organizational skills alongside curriculum differentiation (which may include exemption from studying a foreign language) and assessment arrangements (typically extra time in examinations but also possibly being supplied with a 'reader' or scribe).

Understandably, the teaching of children with dyslexia has focused largely on remediating their reading and spelling problems. However, co-occurring difficulties need to be addressed in their own right. Management programmes should not be limited to literacy instruction, but should also consider the individual child's need for speech and language therapy, occupational/physiotherapy, medication or behavioural programmes for attention deficits and additional maths support.

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Section 5b

Middle Childhood

23

Autism Spectrum Disorders

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INTRODUCTION

Autism spectrum disorders (ASDs) is the term used to describe the range of conditions that share a common core with childhood autism, the prototypic disorder in the spectrum of autistic disorders. All ASDs share the following characteristics:

- *Qualitative impairments in social interaction*, shown by: the use of non-verbal behaviours such as eye gaze and body posture to regulate social interaction; a failure to develop peer relationships; a lack of spontaneous showing and sharing of interests; and a lack of social emotional reciprocity.
- *Qualitative impairments in social communication*, shown by: delayed language development without non-verbal compensation; problems starting/sustaining conversations; repetitive and stereotyped language; and a lack of imaginative and imitative play.
- *Restricted repertoire of interests, behaviours and activities*, shown by: abnormal over-focus on particular topics; adhering to non-functional routines/rituals; repetitive, stereotyped motor mannerisms; and preoccupation with object parts rather than whole.

Sensory abnormalities, including either hyposensitivity or hypersensitivity, and unusual interests in some sensations (e.g. the feel of clothes or the smell of hair) are common. A lack of imaginative play indicates an underlying difficulty with the generation of ideas that is highly relevant in the development of understanding, and thinking about, other people and other situations.

Individuals with impairments that do not meet full criteria for the diagnosis of autism, such as Asperger syndrome, atypical autism or pervasive developmental disorder – not otherwise specified, are currently classified in DSM-IV-TR (*Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition – Text Revision*) and ICD-10 (*ICD-10 International Classification of Mental and Behavioural Disorders in Children and Adolescents*) as having 'pervasive developmental disorders' – though the term 'autism spectrum disorders' is widely used in its place. This group includes a mix of presentations including greater severity in one area than in others, mild impairments in several areas or late onset. The lack of validity for the distinctions between autism-related conditions has led to the proposal that in the forthcoming DSM-5, separate diagnostic categories will be replaced by severity dimensions within one autism spectrum disorder (see Chapter 35).

PREVALENCE

Childhood autism was once considered a rare neurodevelopmental condition, but recent research suggests prevalence rates of 20–40 per 10,000 [1]. Prevalence for the broader autism spectrum may be as high as 100 per 10,000, or 1% [2]. Factors influencing increased recognition of the disorder include: changing diagnostic criteria; more thorough epidemiological studies; recognition that ASD can occur alongside average IQ, and the identification of ASD in those with comorbid disorders or conditions. The prevalence of autism is four

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times higher in boys than girls, and this increases with IQ; in Asperger syndrome the ratio is 10:1 in referred samples. The reasons for this gender difference have not been determined.

ASSOCIATED DISORDERS

Medical conditions of possible aetiological significance have been reported in 10–30% of cases. Figures are lower in population-based samples than in clinically accrued samples, and lower in high-functioning cases than in developmentally delayed cases. The risk of ASD is increased in tuberous sclerosis and in some genetic anomalies such as fragile-X syndrome and Turner syndrome. Tuberous sclerosis may provide an important clue to brain pathology, since lesions in the temporal lobe have been shown to be a risk factor for comorbid ASD. There is a high rate of epilepsy and seizure disorders. Some children develop seizures in infancy, while in others the onset of epilepsy is in adolescence. By adulthood, one-third of individuals with ASD have developed epilepsy.

AETIOLOGY

Autism is a strongly heritable disorder. This genetic predisposition is polygenic in character, with multiple genes responsible, many of likely small effect. None has yet been identified, although considerable international collaborative efforts have indicated that susceptibility genes may be located on chromosomes 2, 7, 16 and 17. It is entirely plausible that the autism phenotype might result from a number of genetic components and mechanisms, and recent evidence has emerged that spontaneous mutations called 'copy number variations' (CNVs) may account for a proportion of cases [3]. The recurrence risk for subsequently born siblings is approximately 5–10%. However, it is now known that milder impairments in social communication skills or language affect up to 10–20% of first-degree relatives, especially those who are male. Familial susceptibility to the 'broader phenotype' has implications for genetic counselling. Non-genetic causation in some cases has not been ruled out, but such instances are probably a small minority. Although a history of prenatal and perinatal obstetric complications is not uncommon, these are likely to be secondary phenomena, consequent on an already abnormal fetus.

In 15–30% of children with ASD there is a period of stasis of development and even a frank loss of skills, most commonly speech (usually before the 10-word stage) [4]. This regression, which is usually noted at 14–20 months, is often accompanied by social withdrawal of the child into 'a world of his own', with less following of others' gaze and a lack of response to speech. Repetitive play behaviours are sometimes noted at this time. There is no explanation why this pattern is present in some children and not others, and the reliance on parental report of behaviour limits knowledge of how widespread such a developmental course is. Later-onset regression following a period of normal development up to 3 years of age or beyond is rare and is referred to as childhood disintegrative disorder.

DIAGNOSIS

Progress has been made in the earlier identification of ASD such that many children, especially those with a more classic presentation of autism in combination with language delay, are identified in the preschool period [5]. Our improved understanding of the emergence of autism in the preschool years stems from recent improvements in screening and study design; prospective studies of genetically at-risk siblings of diagnosed children; and the retrospective analysis of home movies [6]. The utility of early diagnosis stems from evidence that behavioural and social communication interventions can improve outcomes and help parents to understand and better manage their child's behaviour [7].

However, it is still rare for ASDs to be diagnosed before 2 years of age, even though it mainly onsets in infancy and is the result of genetic and other organic factors affecting brain development very early in life. In part this is because abnormalities in behaviours during infancy may be subtle (see Table 23.1). Poor coordination of attention, poor social orienting and play, and a lack of emotional reciprocity may not be noticed before the second year of life. In the high-functioning group (e.g. Asperger syndrome), where language milestones are not delayed and cognitive skills are in the average or superior range, the diagnosis is often not made until school age, or even later. When the affected child is the first, or only child, parents will be less certain about abnormalities of

Table 23.1 Summary of parental concerns that are possible indicators of autism spectrum disorder (ASD) and require further monitoring.

Communication concerns
Does not respond to name
Cannot tell me what he wants
Language is delayed
Doesn't follow directions
Appears deaf at times
Seems to hear sometimes but not others
Doesn't point or wave bye-bye
Used to say a few words but now he doesn't
Socialization concerns
Doesn't smile socially
Seems to prefer to play alone
Gets things for himself
Is very independent
Does things 'early'
Has poor eye contact
Is in a world of his own
Ignores us
Is not interested in other children
Behavioural concerns
Tantrums
Is hyperactive/uncooperative or oppositional
Doesn't know how to play with toys
Gets stuck on things over and over
Toe walks
Has unusual attachments to toys (e.g. is always holding a certain object)
Lines things up
Is over-sensitive to certain sounds or textures
Has odd movement patterns
Absolute indications for immediate further evaluation
No babbling by 12 months
No gesturing (pointing, waving bye-bye, etc.) by 12 months
No single words by 16 months
No two-word spontaneous (and not just echolalic) phrases by 24 months
ANY loss of ANY language or social skills at ANY age

Adapted with permission from Charman T and Baird G. Practitioner Review: Diagnosis of autism spectrum disorder in 2- and 3-year-old children. *Journal of Child Psychology and Psychiatry* 2002;43:289–305.

development during infancy or the early preschool years. Recognition of ASD is an important aspect of differential diagnosis in preschool children presenting with developmental problems.

The particular pattern of symptoms that presents in a 2- or 3-year-old with ASD may differ from that seen at the more prototypic age of 4 or 5 years [8]. In particular, overt repetitive and stereotyped behaviours may be less notable, although when these are seen alongside social and communicative impairments they are highly indicative of ASD.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of autism includes hearing problems, global developmental delay and language delay or disorder, especially receptive language delay. In very young or profoundly delayed children, differentiating autism plus developmental delay from developmental delay alone may be difficult. However, social interest, use of pointing and other non-verbal gestures, imitation and pretend play skills would usually be commensurate with overall developmental level in a child with general developmental delay. In contrast, these are areas that are specifically delayed in children with ASD. Behavioural problems that may present a differential diagnosis of ASD, and are possible comorbidities, include:

- attention deficit hyperactivity disorder (ADHD);
- deficits in attention, motor skills and perception (DAMP);
- oppositional behaviour, including resistance to change;
- acutely anxious behaviour;
- particular fears in certain situations.

Eating and sleeping problems, although common in young children, are seen to a more extreme extent in children with an ASD. Dyspraxic or clumsy children may demonstrate features that overlap with an ASD presentation, and more than 50% of children with ASD will have a problem with coordination.

Approximately 50% of individuals with an ASD have IQs in the intellectual disability range (<70); this percentage has recently been revised downwards from 75% with the increasing recognition of ASD in children with average intelligence.

Typically, non-verbal abilities are considerably better than verbal skills, although this pattern is less clear in high-functioning individuals.

ASSESSMENT AND INVESTIGATIONS

A multidisciplinary approach to assessment is required. The composition of teams varies, but commonly includes a paediatrician, a child psychiatrist, a speech and language therapist, a clinical psychologist and an occupational therapist or physiotherapist.

The information necessary for diagnosis includes a detailed developmental history, parents' descriptions of the everyday behaviour of the child, and direct assessment of the child's social interaction style and communicative and intellectual function [9,10].

- *Observation* of the child's social and communication abilities should take place in structured (e.g. IQ and language assessments) and unstructured (e.g. interactive play assessments) settings. In the unstructured setting it is important to vary the degree of social 'press' in order to elicit spontaneous social overtures, requests and comments and the child's response to and understanding of adult approaches. Another important element, where possible, is the opportunity to observe the child with peers of the same age at nursery/school. This is important because of the difficulty of separating response to familiar and undemanding social routines from the more demanding responses to unfamiliar social presses. The latter require social reciprocity and understanding of the pragmatics of social exchanges, and it is in these situations that the social and communicative impairments that characterize children with ASD will be elicited.
- *Interviews* – the use of structured interview assessments, such as the Autism Diagnostic Interview – Revised (ADI-R), and structured interaction schedules, such as the Autism Diagnostic Observation Schedule – Generic (ADOS-G), help systematize the range and depth of information collected. However, these are time consuming and are not always feasible within the organizational constraints of the clinic, especially when the consultation is led by very specific parental concerns. IQ, language and communication skills and adaptive behaviour should be measured.
- *Physical investigations* may be indicated in some cases, particularly when there is evidence of seizures, if a child has a fluctuating clinical course, or additional features such as motor impairments, e.g. ataxia or loss of skills [9]. Relevant investigations include EEG, screening for fragile-X and chromosomal anomalies, use of a Wood's light to look for white skin patches suggestive of tuberous sclerosis, and a hearing test.

MANAGEMENT AND INTERVENTION

Educational provision should maximize the child's social and communicative abilities, as well as their intellectual functioning and academic attainments. There is increasing evidence that appropriately targeted intervention improves outcome in some children with ASD. There is some evidence of the benefits of intensive early behavioural intervention, particularly when combined with approaches that also target the core deficits in social relating and communication [11]. In addition, there is positive evidence, including from randomized clinical trials, for intervention approaches that emphasize the development of non-verbal social communicative skills in preschool children, including via parent training approaches [12].

Whatever the underlying approach, provision of a structured (and predictable) environment and emphasis on communication skills are important aspects of the preschool and school curriculum for children with ASD. In some, behavioural interventions are needed in order to reduce repetitive, stereotyped, self-injurious and challenging behaviours. Many parents need support, and in both the UK and USA there are web-based national autism societies providing excellent sources of information and services (see 'Sources of support' below). For families with the most challenging children, respite care may be necessary.

Medication

Current pharmacological treatments do not treat the core features of ASD. Neuroleptics such as trifluoperazine and haloperidol have been used to manage stereotyped (including self-injurious) behaviours and hyperactivity but can produce dystonic reactions and drowsiness. Such behavioural problems are usually found in individuals with

Box 23.1 Practice points

- Autism spectrum disorders (ASDs) constitute a diagnostic spectrum of variable severity along at least three dimensions: deficits in social communication, poor language and non-verbal communicative abilities, and circumscribed interests and rigid, repetitive and stereotyped behaviours.
- Diagnosis should rely on information from several sources: a parental report of the developmental history, together with observations of the child undertaking structured social tasks, and in the company of peers. The particular pattern of symptoms that presents in preschoolers may differ from that seen at the more prototypic age of 4–5 years; in particular, overt repetitive and stereotyped behaviours may be less notable.
- The specific genetic and neurobiological basis of autism has not yet been identified and, although it is a highly heritable condition, non-heritable genetic and non-genetic factors may play a role in aetiology.
- Pharmacological treatments are used for epilepsy and for the management of severe behaviour disorders, including self-injury and stereotypies, but their role is relatively a minor one. Appropriate educational placement, behavioural management, social communication interventions and support for parents are the primary management tools. The course and prognosis of autism are variable, and hard to predict until the child is of school age. High IQ and good language ability are the best prognostic signs.

severe developmental delay. Some recent reports suggest that irritability, anxiety, aggression and repetitive behaviours may be ameliorated by the serotonin 2A-dopamine D2 antagonist risperidone, and that parent training in behavioural management provides additional benefits over medication alone [13]. Other studies suggest that autistic symptomatology could be improved by the selective serotonin reuptake inhibitor (SSRI) fluoxetine, but the evidence is weak. Adolescents and adults with Asperger syndrome who have mood disorders may respond well to SSRIs. Clomipramine, a tricyclic antidepressant and 5-HT-uptake inhibitor, has been used to treat obsessional and compulsive behaviours with moderate success.

COURSE AND PROGNOSIS

The course and outcome of ASD are largely dependent on language and IQ, and vary considerably. There is often improvement after the preschool years, especially in the acquisition of language skills. However, for the majority of individuals, significant social impairment is life-long. An IQ above 70 and onset of functional language by 5 years of age are good prognostic signs. Asperger syndrome is associated with a better prognosis because of relatively good IQ and language skills, but only some children with this disorder will be able to make close social relationships outside their family.

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SOURCES OF SUPPORT

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USA – Autism Speaks (http://www.autismspeaks.org/).

24

Somatization and Somatoform Disorders

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WHAT'S NEW

- Demonstration of high levels of anxiety disorder comorbidity [2]
- Description of intergenerational transmission and parental influences [4,5,10,11]
- Replication of the effectiveness of cognitive-behavioural family therapy [12].

INTRODUCTION

Somatic symptoms are common in children and adolescents. In some surveys, young people report a mean of two somatic symptoms (on a 27-item somatization questionnaire) present 'a lot' in the 2 weeks prior to assessment; girls aged 12 years and older have higher scores than similar-aged boys. The most common symptoms are headache, low energy, sore muscles, nausea and upset stomach, back pains and stomach pains. In the general population, 2–10% of children have aches and pains, which are mostly unexplained, and 5–10% of children and adolescents report distressing somatic symptoms or are regarded by their parents as 'sickly'.

Parental reaction

In judging the significance of these symptoms, parents need to decide whether the child is ill, is 'exaggerating' or is upset. Parents are usually aware that worries about school, friendships or family discord may contribute to the symptom, or that symptoms may be used to avoid something the child finds difficult. Common and effective parental reactions are to 'play down' the importance of the symptom, so that the child learns to cope, or to comfort the child and try to find the cause for the distress. There are times, however, when the symptoms become marked and persistent.

Somatization

'Somatization' describes psychological difficulty or distress that is manifested through somatic symptoms, a tendency to experience and communicate somatic distress and symptoms unaccounted for by pathological findings, to attribute them to physical illness and to seek medical help. It is a crucial feature of a number of ICD-10 (*ICD-10 International Classification of Mental and Behavioural Disorders in Children and Adolescents*) and DSM-IV-TR (*Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition – Text Revision*) somatoform and allied disorders, of which the following are most commonly seen in children and adolescents:

- persistent somatoform pain disorder;
- dissociative/conversion disorder;
- chronic fatigue syndrome ('neurasthenia' in ICD-10).

CLINICAL CHARACTERISTICS

Most research on severe, recurrent unexplained pain that leads to medical help-seeking has

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been done on children presenting with stomach aches and headaches, but multiple complaints are common in these children. In a classic study, Apley found that around 10% of schoolchildren had at least three bouts of abdominal pain severe enough to affect activities over a period of 3 months; comparable rates have been found in preschool children [1]. Associated physical symptoms include pallor, vomiting, fever, headache and subsequent sleepiness and lethargy. Children may look quite unwell, which reinforces parental worries about physical illness.

Headaches commonly present as migraine and tension headaches, although in practice the differentiation between the two types is often not clear-cut and both may coexist.

- In migraine the headache is periodic, severe and unilateral, accompanied by a visual aura, nausea, vomiting and a family history of migraine.
- Tension headaches are described as non-paroxysmal, frequent, bilateral, like 'a band' or 'heavy weight' or 'fullness', with associated dizziness.

Abdominal pains and headaches form part of ICD-10's persistent somatoform pain disorder, when the pain is persistent, severe and distressing and occurs in association with emotional conflict or psychosocial problems that are sufficient to be considered to have an aetiological influence. In dissociative (conversion) disorders there is a partial or complete loss of bodily sensations or movements. Symptoms may be brought on by a particularly traumatic event and they tend to remit after a few weeks or months. Pseudoseizures are also seen in children.

In chronic fatigue syndrome (CFS), the main complaint is increased fatigue after mental effort. It is associated with a decrease in occupational performance or coping efficiency in daily tasks and involves:

- difficulty in concentrating;
- dizziness;
- physical fatigue with physical weakness and exhaustion after only minimal effort;
- muscular aches and pains;
- tension headaches;
- sleeping problems;
- worry about decreasing mental and physical well-being.

Functional impairment that is not explained by any associated physical or psychiatric pathology is a key feature of somatoform disorders seen in specialist clinics.

ASSOCIATED FEATURES

Precipitating factors

Somatoform disorders may be precipitated by physical problems and medical treatments; for example, severe abdominal pains may start after an acute gastrointestinal infection. An injury followed by treatment with immobilization may precede loss of sensation or motility in a limb. A flu-type or infectious illness may bring on chronic fatigue syndrome. Stressful events are known to contribute to the development or continuation of problems such as recurrent abdominal pains. Their effect may be mediated by problems in social competence (i.e. child, parent and teacher perceptions of the child's social skills or peer acceptance) in the child and high levels of stress or of physical symptoms in families.

Comorbidity

Between one-third and one-half of children with somatization-related disorders have psychiatric comorbidity [2]; but in severely affected children with chronic fatigue syndrome and in more recent studies of children with recurrent abdominal pain attending paediatric clinics this rises to three-quarters [3]. Emotional spectrum disorders (usually anxiety or depressive disorders) are the most common association. Psychiatric disorders may precede the development of the handicapping functional symptoms, or they develop during its course.

Personality features

These may be relevant precursors. Clinicians have consistently described affected children as conscientious – even obsessional – sensitive, insecure and anxious, and this may be linked to enhanced stress sensitivity in these children.

Box 24.1 Engaging the family in assessment and treatment

- Fully discuss the physical concerns preoccupying the family and the physical tests carried out.
- Address the physiological mechanisms contributing to symptoms when understood (e.g. muscle pains as a consequence of immobilization and abnormal gait; excessive rest may alter sleep).
- Attributing symptoms to being 'all in the mind' is particularly unhelpful. Avoid questioning the child's truthfulness regarding the symptom.
- Families and children are more likely to accept stress and psychological factors contributing to the maintenance of the condition than to its cause.
- Move from the physical to the psychological at a pace the family can cope with.
- Developing strategies to help the child and family cope with the symptoms and reduce the associated functional impairment is the essence of treatment.
- Emphasize that response to treatment may be slow and intermittent but that most children can recover.

Family factors

Family factors are thought to contribute, and a combination of psychological distress and health problems in the family may be specific to childhood somatization. While a genetic contribution is likely – possibly reflecting a biological vulnerability to experiencing different types of symptoms – family ill-health could serve as a model for the child's symptoms, thus sensitizing the child and family and focusing their attention on physical symptoms. Parental emotional over-involvement and reinforcement of the symptoms have been noted [4,5], for example through positive consequences for symptoms, such as allowing the child to stay home from school and giving the child gifts when he or she is sick.

In a few cases, associated problems in the family include profound disorganization, sexual abuse and 'fabricated' illness (e.g. factitious disorder, or Münchhausen by proxy), leading to the child presenting repeatedly for medical assessment and care [6].

Educational concerns

Educational concerns are often noted. The physical problems may start shortly after transfer to secondary school; children may be overconcerned about academic achievement or there may be a history of sensitivity or other problems in social relationships, sometimes including bullying.

DIAGNOSTIC ASSESSMENT

Children with mild disorders will be seen by their general practitioner. Paediatricians and other specialists often see more severe cases. Fewer children attend child and adolescent psychiatric clinics, where assessment needs to include a detailed psychiatric history, mental state of the child and assessment of family function. Psychometry is useful when a mismatch between the child's achievements, capability and expectations is suspected. A detailed history of school attendance is essential.

Even after specialist referral, many families remain anxious about the possibility of an as-yet undisclosed underlying physical disorder accounting for the child's symptoms, and the need for this to be uncovered and treated. The main principles in the assessment of these concerns and in planning treatment are outlined in Box 24.1.

DIFFERENTIAL DIAGNOSIS

Physical symptoms are commonly found in children and adolescents with psychiatric disorders. The following should be considered in differential psychiatric diagnosis:

- school phobia and refusal, where key features are marked fear and avoidance of school;
- anorexia nervosa, with deliberate weight loss and an intrusive dread of fatness;
- depressive disorder, with consistent and persistent lowering of mood;
- anxiety disorders, with prominent, prolonged and persistent feelings of anxiety, worry and restlessness.

Factors that raise suspicion that psychological factors are playing a part in somatic presentations include:

- when stress and physical symptoms occur close together;
- a symptom whose severity is out of keeping with the established pathophysiology;
- concurrent psychiatric disorder;
- the presence of the characteristic child, family and illness factors described above.

REFERRAL TO CHILD AND ADOLESCENT MENTAL HEALTH CLINICS

A psychiatric referral is especially appropriate:

- for diagnostic purposes, when there is uncertainty about the relevance of psychological factors;
- when there is associated psychiatric disorder;
- when major family problems affect the resolution of the symptoms;
- when the child fails to respond to standard paediatric treatment.

PLANNING TREATMENT

Treatment should aim to develop a partnership with the child and family and to adopt a coordinated approach with schoolteachers and other therapists involved. A treatment programme is likely to involve the features outlined in Box 24.2. Weekly therapy sessions at earlier stages may help to identify and review goals.

School

Looking at ways to reduce stresses in discussion with parents and teachers is important. In very

Box 24.2 Features of a treatment programme

- Ascertaining precise daily variations in severity of symptom and associated impairment (diaries can help with this).
- Special techniques to deal with individual symptoms (e.g. distraction techniques, rest periods, relaxation for headaches, analgesics as appropriate, physical exercises for muscular dysfunction).
- Attention to dietary habits and sleep, which may have become altered.
- Acknowledging and addressing as appropriate concerns of the child and family that a rehabilitation programme might make the symptoms worse.
- Modest initial goals to increase normalization of daily activities.
- Gradually increasing daily activities (including school work and attendance), change being set at achievable, consistent, agreed levels.
- Exploring the child's expectations of ultimate goals (e.g. for usual well-being or fatigue levels) as these may be unrealistically high.
- Treatment of associated emotional disorders.
- Management of underlying anxieties.
- Family work.

incapacitated children a period out of school, doing homework only or working with a home tutor, may be indicated. Rehabilitation into school will usually involve gradual and partial reintroduction and a school for 'delicate' children may be required. Some children benefit from admission to a paediatric unit with psychiatric input, or to a psychiatric unit with educational provision.

Family work

This is essential in all cases. The aim is threefold:

- facilitate the parents' engagement in treatment, discussing their ongoing concerns about illness;
- plan a treatment programme and help develop distracting and other coping mechanisms;
- intervene as appropriate for any associated family dysfunction, parental psychopathology or other source of family stress that may impede recovery. Family stresses that were not apparent at initial stages of treatment may become apparent in the course of it.

Drug treatments

Antidepressants can be helpful if there are associated depressive features or because of their sedative effects. Tricyclic antidepressants or fluoxetine, a selective serotonin reuptake inhibitor (SSRI) can be used. Citalopram has proved promising in an open trial. However, the use of SSRIs in children and adolescents is subject to restrictions.

EFFECTIVENESS OF TREATMENT

There have been few satisfactory controlled studies of treatments for childhood somatoform disorders, but clinical reports by hospital paediatricians and child psychiatrists have outlined beneficial effects. Helping parents to understand the links between psychological and physical pain is in itself appreciated by many parents and help make the child's pain less intense and more manageable. The best available evidence of efficacy for unexplained abdominal pain comes from cognitive-behavioural family interventions. Sanders and colleagues [7] in a randomized controlled trial of 7–14-year-olds found that children who had the psychological intervention had a higher rate of complete elimination of pain, lower levels of relapse at 6-month and 12-month follow-up, and lower levels of interference with their activities from pain than children receiving standard paediatric care.

Tension headaches in children and adolescents can be substantially improved by *relaxation training*. In some studies of adolescents this is superior to attention placebo control and equally effective when administered at the clinic and as a home-based self-administered treatment.

Psychiatric admission is indicated if the child is very incapacitated, if there is associated psychopathology and progress is not made with outpatient treatment. Admission may not be accepted by families until psychiatrists have done some facilitating work together with paediatricians.

Legal considerations

In exceptional cases a somatoform disorder will be an expression of severe family disruption and child abuse (including factitious illness). The necessary statutory mechanisms need to be explored to safeguard the child's safety and to allow treatment to proceed. Close cooperation between different professionals involved will be important to avoid different points of view about the condition contributing to protracted investigation and its continuation. If parents are unable or unwilling to engage in any type of treatment for the child, consideration should be given to whether the child's development and safety are being put at risk and care would be better provided away from the family (i.e. by taking the child into care). This is rarely a clear-cut issue and a second opinion from clinicians with special expertise in the area is usually required.

OUTCOME

Clinical reports indicate that the majority of markedly affected children with somatoform disorders who are seen in specialist clinics recover in the short term. Some symptoms persist to a lesser degree in a considerable proportion (about one-third in some studies) and may continue into adulthood (half the subjects in a long-term follow-up study of abdominal pains). Long-term follow-up indicates that these children also have an increased risk for psychiatric disorders in adulthood [8].

PREVENTION

Efforts at prevention might profitably address children of parents with somatoform disorders, young preschool children with functional symptoms [9], and older children with a history of persistent school non-attendance. Once children are receiving treatment, addressing the management of personality features that are likely to render them vulnerable to stress reactions and somatization (e.g. sensitivity to criticism, conscientiousness, proneness to anxiety, as well as excessive academic and behavioural expectations) may help to prevent relapse.

CURRENT CHALLENGES AND FUTURE DIRECTIONS

The psychiatric management of children with somatization and somatoform disorders can be hampered by reluctance by families to entertain a psychosocial contribution to children's symptoms. Hence good liaison between paediatric and psychiatric services is of the essence, and this will be facilitated by further development of paediatric liaison psychiatric teams. Future research into physical and psychological vulnerabilities and into efficacious treatment strategies is called for.

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25

Attention-Deficit Hyperactivity Disorder

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DEFINITION

Attention-deficit hyperactivity disorder (ADHD) is a childhood-onset, impairing, neurodevelopmental disorder [1]. ADHD is a diagnostic category in the *Diagnostic and Statistical Manual of Mental Disorders, 4th edition* (DSM-IV). Hyperkinetic disorder is the diagnostic term used in the *International Classification of Diseases, 10th revision* (ICD-10), and specific criteria are listed in Box 25.1. ICD-10 also includes the category of hyperkinetic conduct disorder. Children with both ADHD and conduct disorder deserve distinction in that they have more severe symptoms of ADHD and a poorer clinical outcome than those with ADHD alone. DSM-IV differs from ICD-10 in several ways; notably it divides ADHD symptoms into two rather than three groups (hyperactive/impulsive and inattention). A diagnosis of ADHD, combined type, requires symptoms in both groups. DSM-IV also allows for diagnosing ADHD, inattentive type and hyperactive-impulsive type.

Comorbidity is common and includes:

- oppositional defiant disorder and conduct disorder;
- developmental problems including reading disability (dyslexia), developmental coordination disorder, speech and language problems;
- tic disorders, including Tourette syndrome;
- anxiety and depression;
- learning/intellectual disability;
- pervasive developmental disorders.

Current diagnostic criteria state that ADHD should be diagnosed in the absence of anxiety disorders, mood (affective) disorders, pervasive developmental disorders and schizophrenia. The co-occurrence of ADHD with these disorders is, however, being recognized and these exclusion criteria may change in ICD-11 and DSM-V, as may the age of onset criterion.

EPIDEMIOLOGY

In the most recent UK epidemiological study [2], prevalence rates were 1.4% for DSM-IV ADHD combined type and 1% for ICD-10 hyperkinetic disorder. Some studies have found higher rates of up to about 5–6%. Although the rates of ADHD recognition, clinician-provided diagnosis and treatment have markedly increased since the 1980s in the UK, USA and Europe, there is no evidence that the prevalence of the disorder or symptoms is rising over time [3]. This suggests that increased rates of treatment are in part due to greater clinician and public awareness of ADHD. Likewise, it has sometimes been assumed that the prevalence of ADHD varies widely in different countries and is especially high in the USA. However, a meta-analysis of worldwide studies found no significant differences between European and US prevalence estimates in ADHD. The results also suggested that reported differences in prevalence appear to be influenced by methodological variation, notably whether or not there is associated impairment,

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Box 25.1 Symptoms of hyperkinetic disorder in ICD-10

Hyperactivity

- Often fidgets with hands or squirms in seat
- Difficulty remaining seated when required
- Runs about or climbs on things excessively in situations when it is inappropriate
- Exhibits a persistent pattern of motor activity (always on the go)
- Often noisy in playing or difficulty engaging quietly in leisure activities

Inattention

- Fails to sustain attention in tasks or play activities
- Often fails to follow through on instructions from others
- Often avoids tasks that require sustained mental effort
- Often easily distracted
- Often loses things that are necessary for tasks or activities
- Appears not to listen to what is being said to him/her
- Fails to pay attention to details, or makes careless mistakes
- Often forgetful in daily activities
- Often has difficulty organizing tasks and activities

Impulsivity

- Difficulty waiting turn in games or group situations
- Often blurts out answers before questions have been completed
- Often interrupts or intrudes on others
- Often talks excessively

Other

- Onset before the age of 7 years
- Impairment/interference
- Pervasiveness of symptoms

the source of information, and higher rates using DSM-IV compared with ICD-10 [4]. Like all neurodevelopmental disorders, boys are more commonly affected than girls. The male:female sex ratio is higher in clinics (7–8:1) than in the community (3–4:1), suggesting that ADHD in females is under-recognized.

AETIOLOGY

Attention-deficit hyperactivity disorder is a complex disorder influenced by the interplay of multiple risk factors. No single risk factor is sufficient to result in disorder. Although there is a strong genetic contribution [5], non-inherited factors are also important. A number of environmental risk factors have been found to be associated with ADHD [1,6], but they have not yet been shown to be definitely causal. Risk factors include:

- *Genetic factors:* ADHD is highly heritable, like autism (heritability around 80%).
- *Specific gene variants:* The most consistent evidence is that dopamine D4 and D5 receptor gene variants are associated. Effect sizes are small. There is replicated evidence for association between a catechol-*O*-methyltransferase (COMT) gene variant and conduct disorder symptoms in ADHD.

- *Genetic syndromes:* syndromes such as fragile X, velocardiofacial syndrome, and tuberous sclerosis may lead to features of ADHD but are rare. Rare subtle chromosomal deletions and duplications (copy number variants), including ones associated with autism and schizophrenia, may also contribute but again do not explain the pathogenesis of disorder on their own. Routine screening of genetic syndromes in non-learning-disabled children is unwarranted.
- *Maternal smoking and stress in pregnancy:* These are associated, but recent evidence suggests that they might not be causal [7,8].
- *Alcohol use in pregnancy:* Exposure to high doses of alcohol results in fetal alcohol syndrome. Evidence that mild alcohol use is important is sparse [7].
- *Low birthweight/prematurity* are associated [7].
- *Extreme early adversity* [9] can lead to features similar to ADHD. It is not known whether milder adversities are important.
- *Environmental toxins:* Lead toxicity and early exposure to pesticides have been considered to be associated with ADHD, besides possibly dietary factors in some children.
- *Family adversity*, notably a negative mother–child relationship, appears to be a consequence of ADHD and seems to improve when ADHD symptoms are treated.

Box 25.2 Key areas of assessment

- Presence of ADHD symptoms as specified in ICD-10 or DSM-IV
- Information from parents and child
- Neurodevelopmental assessment
- Establish degree of impairment and pervasiveness of symptoms
- Information from school or other informants
- Assess for comorbidity and consider differential diagnoses
- Cognitive assessment of the child if learning difficulties suspected
- Physical examination including height, weight and cardiovascular system checks
- Use of rating instruments such as the Conners Rating Scale

ADHD, attention-deficit hyperactivity disorder; DSM-IV, *Diagnostic and Statistical Manual of Mental Disorders, 4th edition*; ICD-10, *International Classification of Diseases, 10th revision*.

COGNITIVE AND NEUROBIOLOGICAL CORRELATES

Routine cognitive testing is not necessary or diagnostically useful. Children with ADHD can underperform on IQ tests. They also show deficits on measures of executive function, response inhibition, prefrontal cortical function, delay aversion, and timing deficits. Structural and functional imaging studies [10] show reduced cerebral, cerebellar and caudate volume, and delayed cortical maturation especially in the prefrontal regions; functional magnetic resonance imaging studies implicate corticostriatal circuit involvement. Animal, genetic and pharmacological studies have implicated involvement of dopaminergic pathways.

DIAGNOSTIC ASSESSMENT

Diagnosis is based on the presence of reported symptoms. The diagnostic process includes a detailed history from the family, observation of the child, and reports from school or other observers (Box 25.2).

Information from parents

This includes a developmental and psychiatric history, exploring symptoms and behaviour in detail. Asking for examples of the behaviour often illustrates the severity of problems and the level of impairment. The clinician should ask the parents to explain what they think could be causing the problems and their attitude to treatment. Finally, completion of rating scales such as the Conners Parental Rating Scale [11] provides baseline information on symptoms and can also be used to monitor treatment response.

Child information and observation

These are important to assess ADHD symptoms and comorbidity, and to consider differential

diagnoses, such as anxiety and mood disorders. The clinician needs to consider the developmental age of the child. Observation while performing tasks that require a certain level of self-control and sustained attention can be helpful but the diagnosis should not be based purely on observed behaviours in clinic. When possible, a school visit can provide invaluable information to support the diagnosis. Teenagers can often describe their symptoms, for instance subjective feelings of restlessness, and provide information on their level of social functioning. However, self-reports of ADHD are not a substitute for reports from informants, have poorer predictive validity than parental reports, and should not be used alone to make the diagnosis [12].

Report from school or other informants

After gathering consent, a report from the school or other informants is crucial. A school report from a teacher who knows the child well provides information on how the child's symptoms and behaviour manifest in a more structured environment, and on academic performance and social relationships. The use of instruments such as the Child ADHD Teacher Telephone Interview (CHATTI) [13] or teacher rating scales (e.g. the Conners Teacher Rating Scale) [14] can be valuable. In young adults who have left school, informant reports still remain an important part of the diagnostic process. A cognitive assessment of the child may be necessary to identify learning disability, but is not routinely required. Some children require assessment by an occupational therapist or physiotherapist if motor coordination problems are noted.

Physical examination

This can be important in ruling out physical causes of the symptoms (e.g. hearing and vision problems). Physical examination needs to include checks on weight, height and the cardiovascular system, especially if medication is later prescribed as part of the treatment plan and the child has a learning disability.

TREATMENT

Current guidance supports the use of multimodal packages of care for the treatment of ADHD; Box 25.3 shows the types of treatment used. Most families benefit from written information about the features and treatment of ADHD, and the addresses of reliable internet websites and voluntary organizations.

Box 25.3 Management of ADHD

Pharmacological interventions

First line:

- Methylphenidate*
- Dexamfetamine*
- Atomoxetine*

Second line:

- Clonidine
- Modafinil
- Bupropion
- TCAs (tricyclic antidepressants)

Psychosocial interventions

- Behaviour therapy including parent training programmes*
- Cognitive–behavioural therapy
- Social skills training
- Family support to reduce stress

School interventions

- Classroom interventions
- Management of general and specific learning difficulties

Related areas

- Elimination and supplementation diets

*Supported by evidence base.

Pharmacological intervention

In preschool-aged children medication is rarely used. Instead, behaviourally oriented parent training programmes are the first treatment option. In cases where ADHD symptoms are severe and there is a marked degree of impairment, and a parenting programme has already been used, medication should be considered. In school-aged children and young adults suffering with ADHD, stimulant medication such as methylphenidate and the non-stimulant atomoxetine have been shown to reduce hyperactivity and improve concentration; these drugs are endorsed by the National

Institute for Health and Clinical Excellence [15]. Although there are short-term therapeutic effects of ADHD medication, there is uncertainty about long-term benefits [16]. Once medication has been started, the child's physical health, including weight and height, and ADHD symptoms need to be monitored regularly and the dosage titrated accordingly (Figure 25.1). The potential advantages of using medication are an initial rapid reduction in core symptoms; this could help children and parents better utilize non-medication strategies, and reduce immediate stress at home and school, which could avoid an escalation of problems leading to school exclusion and family breakdown in the short term. Disadvantages might include an over-reliance on medication alone, side effects, or repeat prescribing over years without consideration of whether there are continued benefits and whether there is the need for additional new non-medication interventions.

Stimulants

Methylphenidate and dexamfteamine are central nervous system stimulants. The mode of action of methylphenidate is not entirely clear, although it appears to result in an increased dopamine concentration in the synaptic cleft by partially blocking the dopamine transporter (DAT). Methylphenidate is rapidly absorbed, reaching maximum plasma levels 1–4 hours after oral administration (see Figure 25.1 for the treatment regimen). Sustained-release formulations with a therapeutic effect of 8–12 hours are also available, making single daily dose administration possible. The main advantage is that the child does not have to take medication at school, reducing problems with storage of tablets and associated stigma for these children. Dexamfetamine also enhances dopaminergic neurotransmission in the central nervous system. The elimination half-life of dexamfetamine allows once- or twice-daily oral administration. The recommended starting dose for treatment of ADHD depends on the age of the child (see Figure 25.1).

Frequent short-term side effects of stimulant drugs include:

- decreased appetite
- sleep disturbance, such as insomnia

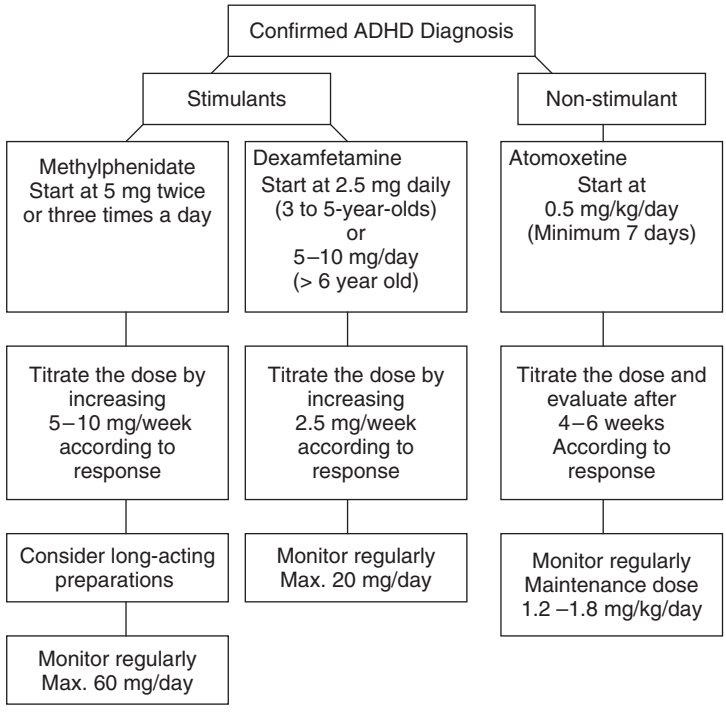

Figure 25.1 Using medication to treat attention-deficit hyperactivity disorder (ADHD).

- headaches
- stomach aches
- drowsiness
- irritability
- tearfulness
- increased blood pressure and pulse.

The effect of methylphenidate on growth in the long term is not clear [17]. The use of drug holidays not only provides the opportunity to assess improvement of ADHD symptoms, but also gives children the opportunity to catch up with their growth by improving their appetite. Finally, the use of stimulant drugs in children with tics and seizures can be considered, but with caution. Stimulants have also been shown to be useful for treating ADHD in children with pervasive developmental disorders and intellectual disability [18], although there is increased sensitivity to side effects in these groups.

Non-stimulants

Atomoxetine is a non-stimulant drug also used in the treatment of ADHD in children aged 6 years and over. The therapeutic effect of atomoxetine is currently considered to be related to the increase of noradrenaline in the cortex through inhibition of presynaptic reuptake. Atomoxetine can be administered as a once-daily dose, although some children benefit from divided daily doses (see Figure 25.1). Common undesired effects of atomoxetine include:

- abdominal pain
- nausea and vomiting
- decreased appetite with associated weight loss
- dizziness
- slight increases in heart rate and blood pressure.

Suicidal thoughts have also been reported to be more frequent among children and adolescents treated with atomoxetine. Finally, although rare, atomoxetine can cause liver damage. Therefore, as with stimulants, regular monitoring of side effects and symptoms is necessary.

Other medications

There is limited evidence supporting the use of other drugs. However, drugs such as clonidine, bupropion, and modafinil have been shown to produce some improvement in ADHD symptoms. Some uncontrolled studies have also suggested the use of tricyclic antidepressants such as imipramine and desipramine. These drugs should be used only as a second-line treatment and when other interventions have not been successful.

Psychosocial interventions

It is recommended that non-pharmacological interventions are also provided to children and adolescents suffering with ADHD. Clinicians must emphasize to parents the need to implement these interventions despite the use of pharmacological treatment. The largest trial to date, the Multimodal Treatment Study of ADHD [19], showed short-term benefits of medication, that adding behavioural treatment reduced the dosage of medication required, but that behavioural treatment alone was not helpful. Children with ADHD may benefit from scheduled activities and regular timetables. Reducing family stress by increasing support, either formally through social services or via voluntary organizations, can be helpful.

Parent training programmes/behaviour therapy are aimed at modifying symptoms and should be considered first for preschool children with ADHD [20]. Parenting packages, such as the Webster–Stratton programme, are behaviourally oriented, usually delivered in groups, and emphasize the use of play, praise, incentives, limits and discipline to improve the child's symptoms and behaviours. Parent training programmes are widely used in CAMHS at present.

Cognitive–behavioural therapy for older children and adolescents can help them to develop a better understanding of their feelings, thoughts and actions, and, theoretically, could improve core ADHD symptoms. Recent work suggests potential benefits for adults who are already being treated with medication [21]. However, to date there is little evidence to support its use in adolescents with ADHD.

Social skills training can be useful to help the child develop socially acceptable behaviours and improve peer relationships. Social skills training includes the use of anger management and problem-solving strategies. At present there is no evidence to suggest that this type of treatment is effective in ADHD.

School interventions

The management of ADHD in school-aged children needs to consider the school setting.

Therefore, training of teachers about the condition and increased support in the classroom is important. Behavioural interventions include class placement, and the promotion of structure and routines in the classroom, and during breaktimes and play activities. Setting small, achievable goals can have a positive effect on self-esteem. Finally, educational psychologists can provide advice on the management of specific educational difficulties and appropriate school placement for children with more complex difficulties.

Other interventions

There has been interest in whether the elimination of certain artificial colourings and preservatives from the individual child's diet can improve the symptoms of ADHD. There is some evidence that this can help a few selected individuals. Some children could also benefit from the use of omega-3 and omega-6 polyunsaturated fatty acids to supplement their diet. However, the evidence base for these interventions is limited at present.

CLINICAL COURSE

Symptoms of ADHD decline with age, but longitudinal studies show that ADHD and associated problems tend to persist into adolescence and adult life [22]. Problems include:

- continuing to meet full diagnostic criteria or showing some symptoms with associated impairment;
- adult ADHD;
- conduct disorder, antisocial behaviour, criminality;
- drug and alcohol misuse;
- educational and employment failure;
- relationship difficulties, driving offences.

Consideration must also be given to the possible effects of ADHD on safe driving in adolescents and young adults.

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26

Challenges in Child and Adolescent Obsessive-Compulsive Disorder

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THE 'HIDDEN PROBLEM'

Young people with obsessive-compulsive disorder (OCD) and their families report that one of the most challenging aspects of dealing with OCD is recognition of the problem. This includes realizing that the symptoms, as they emerge, are part of OCD (Box 26.1).

Box 26.1 Symptoms of obsessive-compulsive disorder

Obsessions

- Intrusive, repetitive, distressing thoughts or images
- Most common themes: contamination, harm coming to others, sexual, aggressive, religious

Compulsions

- Repetitive, stereotyped, unnecessary behaviours
- Most common rituals: washing, checking, repeating, reassurance seeking, ordering

Obsessive-compulsive disorder generally responds to treatment, and the emotional, social and educational disabilities that children may acquire secondary to chronic OCD are likely to be minimized by prompt intervention [1]. Earlier detection and treatment will be promoted by greater public awareness, and there is evidence that early diagnosis and intervention improves long-term outcomes [2]. Fifty percent of adults with OCD remember that their disorder started in childhood, but many failed to get help at this stage, with average delays in diagnosis of 12 years in adults and over 3 years in children [3]. Though paediatric OCD was once considered rare, epidemiological studies have revealed a prevalence of about 1% for OCD [4].

A possible reason for delays in recognizing OCD is the nature of the illness itself. Individuals with OCD usually have good insight, realizing their thoughts and behaviours are unnecessary. They often feel embarrassed by the symptoms and hide them for as long as possible, or may fear they will be asked to stop ritualizing and worry this would be anxiety provoking. Even in a brief primary care consultation, some screening questions such as those in the 7-item Short OCD Screener (SOCS) can be used (available online: http://ocdyouth.iop.kcl.ac.uk/downloads/socs.pdf) [5].

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WHEN DO ORDINARY CHILDHOOD RITUALS BECOME OCD?

Rituals are a part of normal childhood development (e.g. carrying out a bedtime ritual) and should not be confused with OCD [6]. Rituals are common in young children (usually from the age of 2 to 7 years), and parents may fail to notice their child's rituals are becoming more prolonged or distressing. A child's obsessions or rituals may be OCD if:

- the rituals or thoughts upset the child;
- they take up a lot of time (more than one hour per day);
- they interfere with the child's everyday life.

To make a diagnosis of OCD, not only do compulsions and/or obsessions need to be present, but also they must cause functional impairment.

AETIOLOGY

The cause of OCD is not known, but there is increasing research evidence for a biological basis to this disorder [7], although it is highly responsive to psychological intervention.

Family and twin studies support a strong genetic role in the aetiology of OCD, with heritability in children ranging from 45 to 65% [8]; however, the heterogeneity of the disorder complicates the search for specific genes. A promising approach for genetic, imaging and treatment studies is the consideration of OCD dimensions as quantitative phenotypes. OCD is a heterogeneous condition, and factor- and cluster-analytical studies in adults and children have identified four relatively independent symptom dimensions of contamination/cleaning, obsessions/checking, symmetry/ordering and hoarding [6,9].

Brain imaging studies demonstrate differing blood flow patterns in OCD patients compared with controls, and support a frontal-striatal-thalamic model of OCD [10]. Treatment with either medication or CBT is associated with a reversal of functional neuroimaging findings. The neurochemical basis of these differences is not known, but the effectiveness of SSRIs suggests that serotonin is an important neurotransmitter. Glutamate has also been implicated, and trials of glutamate-modulating agents, such as riluzole, in treatment are underway [4,11].

A further finding implicating the basal ganglia in OCD, is that a subgroup of children with OCD may have the disorder triggered by infections. Streptococcal infections trigger an immune response, which in some individuals generates antibodies that cross-react with antigens in the basal ganglia. This subgroup has been given the acronym PANDAS (paediatric autoimmune neuropsychiatric disorder associated with *Streptococcus*) [12,13].

ASSESSMENT OF THE YOUNG PERSON WITH POSSIBLE OCD

Differential diagnosis and identifying OCD

Diagnosing OCD may involve asking the child direct questions about obsessions and compulsions, as these may not be revealed spontaneously. Clinicians need to consider alternative diagnoses, such as depression, other anxiety disorders, developmental disorders that include repetitive behaviours (e.g. autism spectrum disorders) and tic disorders. Psychological instruments may be used to assist with diagnosis and rating of severity. The best validated is the Children's Yale-Brown Obsessive Compulsive Scale (CY-BOCS) [14].

Phenomenology of obsessions and compulsions

Children with OCD usually have both obsessions and compulsions, and an obvious link between them can often be detected, for example contamination fears and excessive washing. However, in some the connection may be less obvious, for example fear of a parent's misfortune if the child does not touch something a certain number of times. Obsessions may not be present; a child may describe that things are just 'not right' if they do not do their ritual. The distressing nature of obsessions can make it difficult for younger children to recognize them as their own thoughts. It is not unusual for younger children to call their obsessions 'voices', and careful examination of the psychopathology is needed to avoid confusing these experiences with psychotic phenomena. Children may have less insight into the irrational nature of obsessions and compulsions, and ICD-10 (*ICD-10 International Classification of Mental and Behavioural Disorders in Children and Adolescents*) and DSM-IV-TR (*Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition – Text Revision*) allow diagnosis of OCD with reduced insight in young people.

Obsessions may cause more distress than compulsions

Particularly in teenagers, predominantly obsessional problems can be difficult to diagnose. The commonest obsessive themes – sexual, aggressive and religious – are often particularly embarrassing to disclose. It is helpful to enquire about these directly, and explain that everyone has intrusive thoughts. It is important to explain that an obsessive thought should not be confused with an impulse to act, and in no way reflects the nature or behavioural leanings of the person experiencing it. This is especially important when someone has socially concerning obsessions, such as having sex with children. If these recurrent thoughts are not recognized as an obsession (which does not imply an actual wish to act on this intrusive thought), an inappropriately high level of risk may be inferred, when there is none.

Secondary risks, the unintended consequences of acting on compulsions or avoidance, should be considered. For example, a person with contamination fears about food may restrict their dietary intake. Also, without effective treatment, harm may occur in individuals whose OCD is severe during critical phases of development; for example, withdrawal from school due may mean they also miss out on important educational and social developmental experiences [15].

Comorbidities

Consistent with adult studies, approximately 80% of young people with OCD have at least one comorbid psychiatric disorder [16]. The commonest comorbidities in children are attention deficit hyperactivity disorder (ADHD; 34–51%), depression (33–39%), tics (26%), specific developmental disabilities (24%), oppositional defiant disorder (ODD; 17–51%) and anxiety disorders (16%) [4]. Careful assessment is required both to distinguish symptoms of OCD from other disorders and because some comorbidities may influence response rates to particular treatments [17–20].

TREATMENT

Helping parents and young people become informed about OCD is crucial, and there are several good publications available (see 'Further reading' below). The UK National Charity, OCD Action, provides information and links to support groups. For many families, knowing they are not alone is the first step towards recovery.

There are two interventions of proven efficacy: cognitive–behavioural therapy (CBT) with exposure and response-prevention (ERP), and specific medication, which can be used independently or in combination. One randomized controlled trial (RCT) has directly compared the efficacy of CBT and SSRIs (sertraline), and showed no significant difference between them, whilst combination therapy was more effective [16]. A meta-analysis of RCTs found that whilst both CBT and medication are effective treatments for paediatric OCD, CBT is associated with greater effect sizes than pharmacotherapy. Pooled effect sizes were 1.45 and 0.48 respectively [21]. There is no evidence that psychoanalytic psychotherapy is of benefit for OCD.

In the UK, the National Institute for Health and Clinical Excellence (NICE) recommends a 'stepped care' model, with increasing intensity of treatment according to clinical severity (Figure 26.1) [22]. The assessment of the severity and impact of OCD can be aided by the use of the CY-BOCS [14]. CBT incorporating ERP is recommended as first-line treatment in children and adolescents, with the subsequent addition of an SSRI, then consideration of clomipramine and augmentation strategies with atypical antipsychotic medications as necessary. Admission to hospital is rarely indicated, but may be needed if there is risk to safety or physical health.

Young people becoming experts: causes of OCD

It is helpful for patients and families to have appropriate information about our current knowledge of the aetiology of OCD. Understanding that OCD is a neurobiological disorder helps young people and families realize it is not their fault. It is also helpful to stress that treatments really work, and that many successful people have coped with OCD.

Cognitive—behavioural therapy (CBT)

Studies in adults and young people have shown CBT to be an effective treatment for OCD, with 40% to 88% of young people achieving remission. Studies have shown that CBT for paediatric OCD can also be effectively delivered in groups or by telephone [20,21,23].

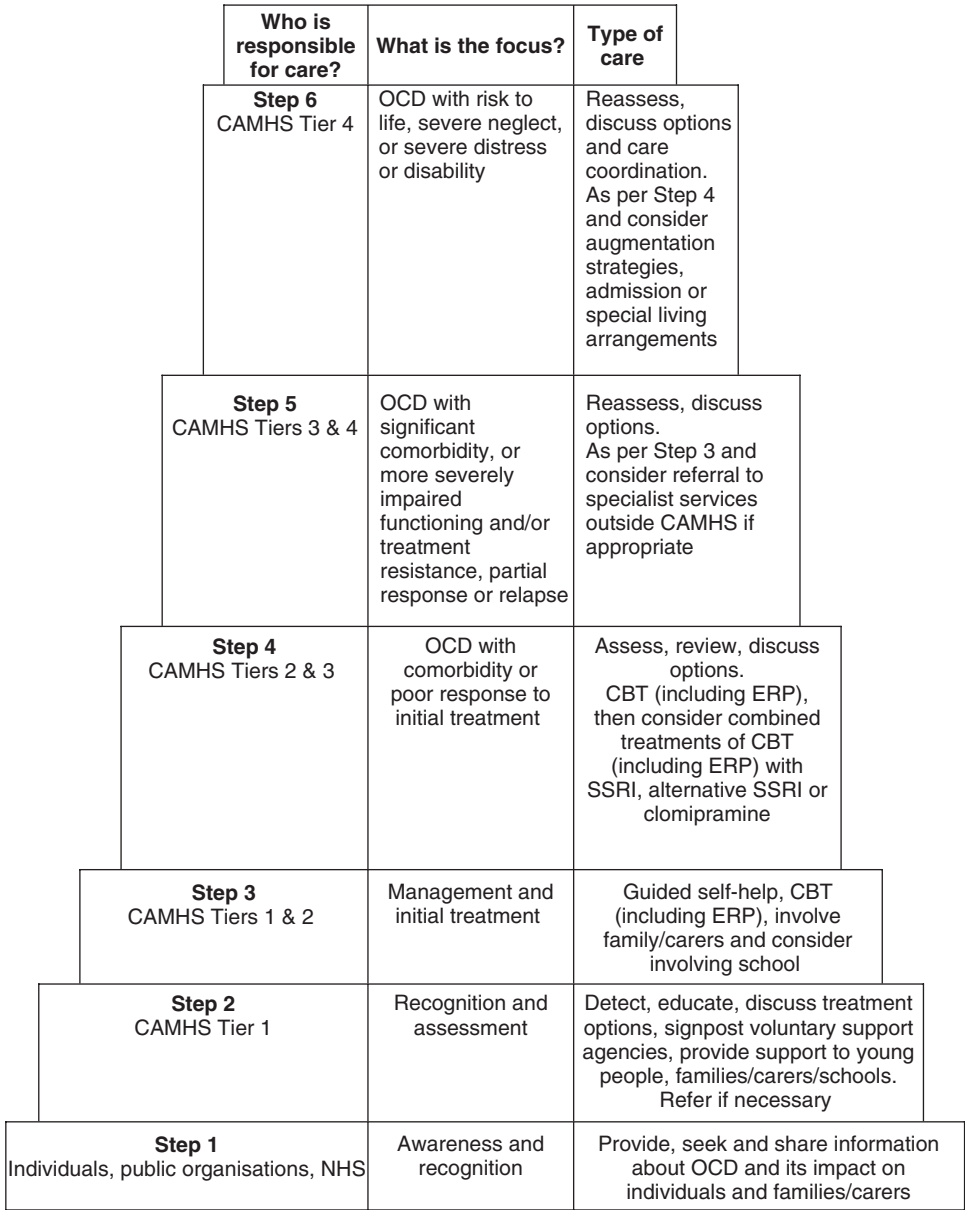
Who is responsible for care?
What is the focus?
Type of care
Step 6
CAMHS Tier 4
OCD with risk to life, severe neglect, or severe distress or disability
Reassess, discuss options and care coordination. As per Step 4 and consider augmentation strategies, admission or special living arrangements
Step 5
CAMHS Tiers 3 & 4
OCD with significant comorbidity, or more severely impaired functioning and/or treatment resistance, partial response or relapse
Reassess, discuss options. As per Step 3 and consider referral to specialist services outside CAMHS if appropriate
Step 4
CAMHS Tiers 2 & 3
OCD with comorbidity or poor response to initial treatment
Assess, review, discuss options. CBT (including ERP), then consider combined treatments of CBT (including ERP) with SSRI, alternative SSRI or clomipramine
Step 3
CAMHS Tiers 1 & 2
Management and initial treatment
Guided self-help, CBT (including ERP), involve family/carers and consider involving school
Step 2
CAMHS Tier 1
Recognition and assessment
Detect, educate, discuss treatment options, signpost voluntary support agencies, provide support to young people, families/carers/schools. Refer if necessary
Step 1
Individuals, public organisations, NHS
Awareness and recognition
Provide, seek and share information about OCD and its impact on individuals and families/carers

Figure 26.1 NICE (National Institute for Health and Clinical Excellence) stepped-care model for obsessive-compulsive disorder (OCD) in children and adolescents [22]. CAMHS, child and adolescent mental health services; CBT, cognitive–behavioural therapy; ERP, exposure and response-prevention; SSRI, selective serotonin reuptake inhibitor. Adapted from NICE Guideline 31, The Stepped-Care Model 2005.

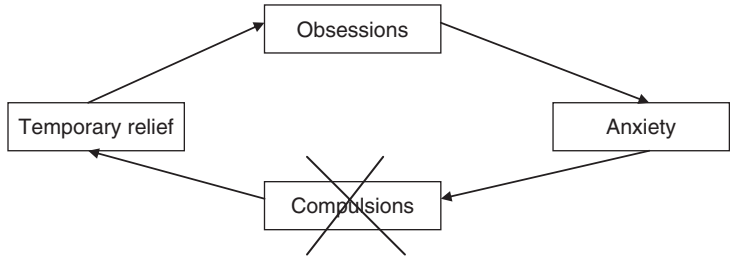

Figure 26.2 The 'vicious cycle of obsessive-compulsive disorder (OCD)'. Obsessions generate anxiety, whilst compulsive rituals are performed to reduce the anxiety, but in fact this produces only temporary relief and reinforces the cycle. Exposure and response-prevention (ERP) acts by exposing a young person to the feared situation whilst resisting compulsions, in a graded manner, to break this cycle.

Anxiety: A key aspect of overcoming OCD and engaging in psychological treatment is understanding the role of anxiety in OCD. Education about anxiety forms a starting point for any successful cognitive–behavioural programme (Figure 26.2).

OCD as an 'intruder': Child-oriented manuals for treating OCD commonly use an externalizing approach [24]. The child is helped to see OCD as an 'intruder' who is spoiling their life by seeking to control their thoughts and actions. Treatment is aimed at giving the child and family effective means of controlling the intruder – saying 'No' to OCD. This 'externalizing' approach to the disorder, giving it a name, and learning how to 'fight' it, is a useful technique in child and adolescent treatment. *The Secret Problem* is a helpful children's cartoon book about OCD that exemplifies this approach [25].

Exposure and response-prevention: CBT protocols are generally based on ERP: 'exposure' (facing up to the feared stimulus) and 'response-prevention' (resisting the urge to carry out a ritual in these circumstances) [24]. In adult and child studies, ERP appears to be a critical therapeutic component. Cognitive protocols, which tackle underlying beliefs about connections between thoughts and behaviours, are also being evaluated [26].

It helps to explain to children that the first step of CBT involves a detailed assessment of the problem, often starting with keeping a symptom diary. Resisting compulsions makes OCD sufferers very anxious, and it is important for them to learn how to confront this anxiety. Step-by-step exposure to the anxiety-provoking situation, while resisting the urge to respond with compulsive behaviour, shows the patient that their anxiety level steadily decreases under these conditions, and they realize that compulsive behaviour is not the only way to relieve their anxiety. The young person designs the treatment programme with the therapist, so that little by little they can overcome their fears. 'Accommodation', or the participation by family members in OCD rituals, is common, elicited by their child's distress and unwittingly reinforcing the OCD, and is reported in up to 75% of parents [27]. Families need to learn about OCD, and how to help their child fight back [27,28]. CBT is not usually a long treatment; most children who respond to this approach do so in 8–12 sessions.

Medication

There is extensive clinical trial literature in adults and young people with OCD demonstrating efficacy for the serotonin reuptake inhibitor (SRI) group of antidepressants. Clomipramine, an SRI, remains a useful drug for some, although its side-effect profile (sedation, dry mouth, cardiac side effects) make it generally less acceptable than selective serotonin reuptake inhibitors (SSRIs), which are now the first-line medication. All SSRIs appear to be equally effective, although they have different pharmacokinetics and side effects [29]. There has been concern about the use of SSRIs in depressed youths, with meta-analyses suggesting low levels of efficacy and an increase in behavioural

activation including suicidal behaviours, although recent evidence is more positive. In contrast, SSRIs appear effective in paediatric OCD, with 'numbers needed to treat' (NNT) ranging from 2 to 10, and there is no significant evidence of increased suicidality [30,31]. However, given recent concerns there should be close monitoring for side effects.

Only sertraline and fluvoxamine are currently licensed for use in children in the UK, but other SSRIs could be used 'off-label' by a specialist in exceptional circumstances. For example, in comorbid depression, SSRIs should be used with caution, and the only one not currently contraindicated in depressed youth is fluoxetine.

About 70% of individuals with OCD respond favourably to medication, and if a first SSRI is ineffective or not tolerated, it is worth trying a second. Both clomipramine and SSRIs have a delayed onset of action, with full therapeutic effects not being apparent for 8–12 weeks. It is therefore worth waiting for a response at a moderate therapeutic dose, rather than moving rapidly to high doses, which will increase the chances of side effects. The optimal dose is variable, and should be determined by titrating from a low starting dose. The effective doses needed for adult OCD seem to be higher than for depression, and this may also be the case in younger people.

PROGNOSIS AND ONGOING CARE

Early detection and treatment is likely to minimize secondary disabilities and continuation into adulthood, but as yet, few assertively treated early-onset cohorts have been followed up long term. Long-term follow-up studies thus far have found a persistence rate of 40%, and further studies into predictors of outcome and augmentation strategies are needed [32,33]. If a young person has responded to medication, treatment should continue for at least 6 months after remission [22]. CBT should give individuals strategies for dealing with transient symptom recurrence, and theoretically obviate the need in some people for long-term medication. Many people with early-onset OCD respond to treatment and lead fully functioning lives. It is important that throughout their lifespan, people with OCD should have access to support as needed, and NICE recommends that if relapse occurs, they should be seen as soon as possible, rather than placed on routine waiting lists.

Box 26.2 Key learning points

- Obsessive compulsive disorder (OCD) is a neurobiological disorder characterized by obsessional thoughts and compulsive behaviours leading to functional impairment.
- It is common in young people; however, delays in diagnosis can occur.
- Without treatment, OCD can become severe and chronic; however, effective evidence-based treatments exist: cognitive–behavioural therapy (CBT) with exposure and response-prevention (ERP) alone, or in combination therapy with selective serotonin reuptake inhibitors (SSRIs).

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A comprehensive account of recent research and its impact on treatment strategies and choices.

Derisley J, Heyman I, Robinson S *et al*. (2008) *Breaking Free from OCD. A CBT Guide for Young People and Their Families*. London and Philadelphia: Jessica Kingsley Publishers.

March JS and Benton CM. (2007) *Talking Back to OCD: the Program that Helps Kids and Teens Say "No Way" – and Parents to Say "Way to Go."* New York: Guilford Press.

Both of these are CBT self-help books that young people could work through alone, or ideally with a parent or therapist.

Wagner A. (2002) *Up and Down the Worry Hill: a Children's Book About Obsessive-Compulsive Disorder and its Treatment*. New York: Lighthouse Press.

An illustrated book designed to help parents and professionals explain OCD to younger children through the story of 'Casey', a young boy with OCD.

Waltz M. (2000) *Obsessive Compulsive Disorder: Help for Children and Adolescents*. Patient Center Guides.

Written by a woman with OCD who has two children with OCD, this book provides information about OCD, its diagnosis and treatment and advice for working with schools.

Wells J. (2006) *Touch and Go Joe. An Adolescent's Experience of OCD*. London: Jessica Kingsley Publishers.

A revealing insight into the life of a teenager with OCD.

USEFUL WEBSITES

OCD youth: www.ocdyouth.info
OCD Action: www.ocdaction.org.uk
International OCD Foundation: www.ocfoundation.org

27

Anxiety Disorders in Children and Adolescents

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Anxiety is an unpleasant feeling of tension or apprehension accompanied by physiological changes and worries or fears. It can become maladaptive if excessive or developmentally inappropriate; if it also causes significant functional impairment, it can be considered to be an anxiety disorder.

It is important to bear in mind developmental differences in the presentation of normal anxiety and anxiety disorders (Table 27.1). Cognitive skill endows adolescents with the capacity to imagine and ruminate on increasingly complex and abstract threats. What is seen as normal for a young child may be considered a disorder in an older child.

DIAGNOSTIC FEATURES

Children with anxiety disorders show a range of symptoms, from mild distress to incapacitating anxiety. ICD-10 (*ICD-10 International Classification of Mental and Behavioural Disorders in Children and Adolescents*) requires the presence of a number of physiological changes occurring either in a specific feared situation or for a specific duration. Making an early diagnosis is important: many anxiety disorders remain untreated in the community, causing distress and impeding academic and social functioning. Failure to diagnose may interfere with the child's acquisition of social skills and result in social dysfunction.

Separation anxiety disorder is an excessive and/or developmentally inappropriate anxiety about separation from attachment figures or excessive worrying about the figure's welfare. Impairment might include school refusal. ICD-10 criteria include an onset before 6 years old and a duration of at least 4 weeks.

Generalized or over-anxious anxiety disorder is characterized by excessive worry and anxiety, which is generalized and persistent, and not restricted to any particular situation or object. This 'free-floating' anxiety is hard to control and is frequently accompanied by a more restricted set of somatic complaints than those found in adults, including restlessness, fatigue, muscle tension and sleep disturbance. These children may show concerns about their competence, excessive self-consciousness, and a strong need for reassurance.

Social phobia involves the marked, persistent fear of embarrassment in social situations involving exposure to unfamiliar people or to scrutiny;

Table 27.1 Fear, and its typical developmental stages.

Age	Fears
9 months to 3 years	Separation, strangers
3–6 years	Animals, darkness, 'monsters'
6–12 years	Performance anxiety
12–18 years	Social anxiety
Adulthood	Illness, death

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these situations are then usually avoided, thereby reinforcing the anxiety associated with them and leading to social isolation. When confronted with a phobic situation (e.g. public speaking or going to a party) the adolescent will experience anxiety symptoms, and at least one of: blushing, shaking, or fear of vomiting, micturition or defecation.

Panic disorder involves recurrent and unexpected attacks of severe anxiety that are not restricted to any particular situation (Box 27.1). The young person may show persistent apprehension about future attacks (anticipatory anxiety) or its feared implications (e.g. losing control, having a heart attack).

Box 27.1 Characteristic symptoms of panic disorder

A discrete period of intense fear or discomfort that develops acutely and is associated with multiple physical symptoms, including:

- palpitations
- sweating
- trembling
- shortness of breath
- chest pain
- nausea.

Specific or simple phobias are characterized by excessive fears of discernible, circumscribed objects or situations that provoke an immediate anxiety response. In children this may be manifested as crying, tantrums, freezing or clinging. Adolescents may recognize that the fear is excessive. Particularly significant for medical practice are phobias of injections and medical procedures.

EPIDEMIOLOGY

Anxiety disorders are one of the most prevalent categories of psychopathology in children and adolescents (Table 27.2). At least one-third of children with anxiety disorders meet the criteria for two or more anxiety disorders. General comorbidity with other psychiatric disorders – including oppositional defiant, depressive, hyperkinetic disorders, and substance abuse – is 40%. Muris *et al.* [1] found that 84% of their sample of children and adolescents with pervasive developmental disorders had anxiety disorder.

AETIOLOGY

Temperament

Longitudinal studies suggest that anxiety disorders tend not to arise de novo but build on pre-existing temperamental traits. Examples include: behavioural inhibition (showing fear and withdrawal in unfamiliar situations and high sympathetic reactivity), passivity, shyness and an anxious-resistant attachment style.

Genetics

Family and twin studies show that genetic factors play a significant role. These disorders tend to run in families but with little specificity, except for panic disorder.

Table 27.2 Epidemiological characteristics of anxiety disorders in children and adolescents.

Disorder	Prevalence	Age of onset	Sex ratio
Separation anxiety disorder	2–4%	Pre-puberty; peaks at 7 years	Approx. equal
Generalized anxiety disorder	3%	Increased incidence in adolescence	Approx. equal
Panic disorder	5%	Late teens	Approx. equal
Social anxiety disorder	1–7%	11–15 years	Commoner in girls
Specific phobia	2–4%	> 5 years	Equal

Table 27.3 Information processing biases: threat attention and threat appraisal.

Process	What is it?	Bias in anxiety disorder	Relative specificity to anxiety disorder subcategories
Threat attention	Attention is rapidly directed towards environmental threats	Tendency to allocate attention automatically towards threats As threat intensity increases, a tendency to avoid the threat develops	Low
Threat appraisal	Events are interpreted as meaningful and threatening to the individual	Children with anxiety disorders exhibit a reduced threshold for classifying stimuli as dangerous	High E.g. adolescents with social phobia exhibit a threat appraisal bias specifically for social stimuli

Neurobiology/neuropsychology

Pine has constructed a neuropsychological model of childhood anxiety [2]. Neuroimaging studies, mostly in adults, have demonstrated amygdala–prefrontal circuitry abnormalities, areas well known for their role in memory, learning and emotional regulation. Such biological changes are associated with information processing biases, namely threat attention, threat appraisal and fear conditioning (Table 27.3).

Parent–child interactions

Retrospective and observational studies have found that parents of anxious children have an excess of controlling and/or rejecting styles of child-rearing, high 'expressed emotion' with emotional over-involvement towards their children. It is unclear, however, whether the parenting style contributes to the child's anxiety, or vice versa. It is possible that parental behaviour of this type impedes the development of autonomy, so that the child feels less safe and more anxious. Parents with anxiety problems who feel threatened themselves may promote the perception of threat in these children and impede the development of coping skills; children may therefore develop anxiety problems via modelling.

Catastrophic life events

These are related to post-traumatic stress disorder. Other anxiety disorders may be related to adverse life events, particularly those characterized by threat or loss, such as a death or break-up in the family.

Social adversity

Parents' emotional availability and ability to help contain their children's anxieties and fears may be affected if they are having to deal with multiple social problems. Children living in families where the parents are facing chronic stressors such as overcrowding, poverty and marital discord are more likely to experience insecurity and feel anxious and fearful.

ASSESSMENT

A diagnostic assessment involves addressing the possible aetiological factors and seeking information from several perspectives (as reports of anxiety symptoms may differ with different informants). One should bear in mind developmental differences in the presentation of anxiety disorders, as compared with transient, developmentally appropriate fears; assessment also needs to consider undiagnosed learning

disability. Differential and comorbid diagnoses include: autistic spectrum disorder, oppositional defiant disorder, attention deficit hyperactivity disorder (ADHD), depression, alcohol abuse and post-traumatic stress disorder.

Medical assessment should include a thorough medical history and physical examination, excluding disorders (e.g. hyperthyroidism, arrhythmias, epilepsy, caffeinism) and drugs (e.g. steroids, sympathomimetics) that can mimic or provoke anxiety states.

PROGNOSIS

The prognosis of anxiety disorders depends on: comorbidity, age of onset, increased severity at baseline, and type of disorder.

The highest one-year remission rates occur in separation anxiety disorder (almost all children), the lowest in panic disorder (less than 75%) and in more severely affected children. Many children develop new psychiatric disorders at follow-up (often new anxiety disorders) and in adulthood. Data from a community epidemiological study showed that different types of anxiety disorder in childhood predicted anxiety and other psychiatric disorders in adolescence; the only exception was generalized anxiety disorder, which predicted only conduct disorder [3].

Although most adolescent anxiety disorders do not persist into adulthood, most adulthood disorders are preceded by an anxiety disorder in adolescence. Moreover, anxiety disorders of childhood lead to a 2–5-fold increase in anxiety disorders, depression, suicide attempts and psychiatric admissions in later life. They are associated with increased rates of alcohol and substance abuse and smoking, possibly as a means of self-medicating. In adults, anxiety disorders are linked to an increased risk of academic failure, low-paid employment, dependence on state benefits, and reduced quality of life.

TREATMENT

Even though anxiety disorders are common in childhood, affected children often do not receive treatment. Treatment may involve a combination of approaches, the type of which should depend on the ever-evolving evidence base, as well as on the individual case. For example, patients with specific phobias are more likely to be offered behavioural treatment, whilst comorbid family dysfunction may require family therapy. The preference of the child and/or family and the resources available may also influence the choice of treatment. Although the UK's National Institute for Health and Clinical Excellence (NICE) developed national guidelines on anxiety disorders in 2004 and 2007, these pertain only to adults.

The main principles of treatment should include stress reduction, education about the nature of anxiety, improving coping mechanisms, and engagement of the family to help support changes. Parents may need to resolve their own problems related to separation and anxiety to avoid exacerbating the child's symptoms.

Behavioural therapy and cognitive—behavioural therapy

Behavioural therapy models derive from (classical and operant) conditioning, social learning, and information processing theories. Behaviour therapy can target the child's behaviour in the context of home and school. Specific techniques include:

- systematic desensitization and exposure for specific phobias, including school phobia;
- relaxation training;
- modelling of appropriate behaviour;
- role playing;
- rewards for desirable behaviour.

Cognitive–behavioural therapy (CBT) combines a behavioural approach (e.g. exposure) with cognitive techniques (e.g. positive self-statements) aimed at altering cognitions and behaviour. The child is asked to reframe his or her thoughts in a more positive way, which in turn alters behaviour. Children aged 10 years and above can benefit from cognitive techniques. Various books, such as the *Think Good, Feel Good* series, provide accessible cognitive–behavioural material for the clinician and patient alike [4]. A recent meta-analysis demonstrated CBT's efficacy in childhood anxiety disorders and found little difference in effect size between short (<10 sessions) or long, group or individual CBT; this might have implications for cost-effectiveness [5].

Other individual psychotherapies

Psychodynamic psychotherapy focuses on underlying fears and anxieties. Although a strong evidence

base is lacking, a prospective outcome study and a retrospective case review documented good outcome in a number of children, especially those who underwent more sessions, were younger, and had phobic symptoms.

Family therapy

If the child's symptoms are seen as a sign of family dysfunction, family therapy may work with the family to help change dysfunctional patterns of interaction and thus reduce the child's anxiety symptoms.

Pharmacotherapy

Medication can be used, although rarely in isolation. It is more commonly an adjunct to a comprehensive package of care, including psychological techniques for symptom management, as this may help prevent relapse after medication has been discontinued. Medication should be considered in older children and adolescents with more severe symptoms, bearing in mind side-effect profiles and comorbidity.

Antidepressants: These are the most commonly prescribed medication, although there is a smaller evidence base in children than in adults. Tricyclic antidepressants have previously been prescribed, but selective serotonin reuptake inhibitors (SSRIs) are now favoured due to better side-effect profiles and because they are relatively safe in overdose. Fluoxetine and fluvoxamine have been shown to be effective in the treatment of children and adolescents with social phobia, generalized anxiety disorder and separation anxiety disorder [6].

Two studies showed that sertraline was effective and generally well tolerated in patients with generalized anxiety disorder [7,8]. Walkup *et al.* also included patients with separation anxiety and social phobia and demonstrated the added efficacy of combining pharmacotherapy with CBT; however, the combined therapy group was not double-blinded [8].

In studies of depressed children and adolescents, SSRIs have been associated with suicidal ideation and non-fatal acts, and the Medicines and Healthcare products Regulatory Agency (MHRA) (and NICE in the UK) have recommended that only fluoxetine has a favourable risk–benefit profile for depression. The risk of such side effects in non-depressed patients is uncertain, although it is important to be mindful of the frequent comorbidity of anxiety with depression. In the UK, no antidepressants are licensed for the treatment of children with anxiety disorders, but fluvoxamine and sertraline are licensed for use in children with obsessive-compulsive disorder. The evidence base and guidelines on the use of antidepressants in children are ever-evolving, and the reader is urged to consult the latest reports.

Other medication: Benzodiazepines are generally not recommended in children: double-blind controlled trials have failed to demonstrate efficacy, and behavioural disinhibition is a risk. There are few data on beta-blockers. Case reports and open trials have shown a reduction in anxiety symptoms after buspirone treatment, although it can be associated with disinhibitory reactions and aggression.

PREVENTION

Given the high prevalence rates and negative consequences of anxiety disorders, as well as the clinical and public concerns over the use of medication, cost-effective prevention measures would be a welcome strategy. Prevention interventions are well placed to target multiple risk factors simultaneously; this is important given the complex interplay between risk factors and the multiple pathways of development for psychological disorders. Farrell and Barrett have reviewed current practice in prevention research in anxiety and depressive disorders, and described an evidence-based cognitive behavioural programme run in Australian schools [9].

CONCLUSION

Anxiety disorders in children and adolescents are relatively common, but can be disabling and persist into later life. They may first present to the paediatrician or general practitioner. It is therefore important for all professionals who deal with children and adolescents to be aware of possible manifestations of anxiety in this age group. Early differentiation between anxiety symptoms and anxiety disorder facilitates appropriate mental health referrals for further assessment and treatment, enabling the child or adolescent to return to normal functioning.

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28

Childhood Behavioural Disorders

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INTRODUCTION

Childhood behavioural disorders, including oppositional defiant disorder (ODD) and conduct disorder (CD), continue to represent the most commonly presenting disorders at community child and adolescent mental health services [1,2]. Concern regarding childhood deviance is not a recent development. Since the time of Plato, societies have struggled with how to understand and manage the behaviour of out-of-control children, with ongoing debate as to the responsibility and culpability of children for their actions [3]. However, there is evidence that such disorders have become significantly more common over the last 25 years [4], and this is reflected in the increasing media and public concern around such behaviours.

DEFINITION

Oppositional defiant disorder refers to a pattern of negative, hostile and defiant behaviour that is clearly outside the normal range of behaviour for a child of the same age and sociocultural context. These behaviours include a variety of features such as often losing one's temper, arguing with adults, refusing to comply with adults' requests, often being angry and spiteful, and deliberately annoying others. Such children do not present with more severe dissocial or aggressive acts [5].

In contrast, CD involves a repetitive and persistent pattern of behaviour in which the basic rights of others or major age-appropriate societal norms are violated. Such behaviour includes aggression to people and animals, destruction of property, deceitfulness or theft, and serious violations of rules such as often staying out overnight or running away from home [5].

SUBTYPING

Childhood behavioural disorders are categorized in different ways in the two major diagnostic systems. In the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition – Text Revision* (DSM-IV), they are divided into childhood onset (before age 10 years), adolescent onset (after age 10 years) and ODD [5]. In the *ICD-10 International Classification of Mental and Behavioural Disorders in Children and Adolescents* (ICD-10), similar behavioural disorders are classified as socialized, unsocialized, confined to the family context, and ODD, reflecting the social context in which these behaviours occur [6].

There has been considerable debate regarding whether or not ODD and CD are distinct disorders or simply represent a continuum of increasingly challenging behaviours. Although DSM-IV excludes a diagnosis of ODD in the presence of CD, there is considerable symptom overlap between the two groups. Most research supports the distinction of oppositional behaviour and covert delinquent behaviour, but it is not yet clear whether aggression should be grouped with either or represents a separate category of its own [7]. Whilst some children will grow out of ODD, in others it can be a precursor of CD, which in turn can lead to the later development of antisocial personality disorder [7].

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One of the reasons for subtyping conduct disorders is to try to separate those young people who are likely to grow out of their behaviour problems from those who persist with such behaviours into adulthood. DSM-IV refers to the number and intensity of symptoms as clinical indicators of severity. Other relevant factors include the presence of overt (confrontational, such as fighting) versus covert (hidden, such as theft) symptoms, comorbid mental health disorders, particularly ADHD, and early symptoms of antisocial personality disorder [7].

The presence of callous-unemotional traits, characterized by a lack of empathy for others, lack of guilt and inability to express emotions to others, does appear to signify a distinct subgroup of young people with a more extreme pattern of aggressive behavioural problems [8].

EPIDEMIOLOGY

As with many epidemiological studies of child mental health disorders, prevalence rates for childhood behavioural disorders are difficult to quantify due to changing diagnostic criteria and methodological variations in study design. In 1975 Rutter *et al*. compared the rates of child mental health disorders in different settings within the UK, showing a prevalence rate for CD of 4% for rural populations, increasing to 9% in urban centres [9]. Many studies have shown CD to be more common in boys than in girls. In more recent UK-based community surveys, conduct disorders occur in 6.9% of primary school-aged boys and 2.8% of girls, whilst in secondary school-aged children these prevalences rise to 8.1% and 5.1% respectively [10].

There is also evidence of a clear increase in the prevalence of CD with age, with boys showing a linear rise from an early age throughout childhood, whilst girls show a different pattern, with rates increasing in adolescence [11]. However, different subsets of behaviours appear to have differing epidemiological trends. Whilst serious physical aggression and rule violations rise in adolescence, lesser forms of aggression, such as fighting with peers, decline with age [11].

Childhood behavioural disorders often appear to show some longevity of symptoms, with 40% of 7–8-year-olds with CD becoming young offenders in later life, whilst over 90% of such offenders have a history of CD as children [12]. Those children who develop CD at an earlier age will often have a worse prognosis than those who develop such problems in adolescence [13].

Epidemiological studies also show the relationship between childhood behavioural disorders and other mental health disorders including ADHD, depression and anxiety [13]. More than one-third of girls and almost one-half of boys with ODD or CD present with a comorbid non-antisocial disorder [11]. The presence of ADHD is particularly found to influence the development, course and severity of CD. Young people with CD and comorbid ADHD have a much earlier age of onset of disruptive behaviour than those with CD alone [14].

AETIOLOGY

The development of childhood behavioural disorders incorporates a broad and complex range of biological and psychosocial risk factors.

Biological factors, including genetics, have long been implicated in the development of childhood behavioural disorders, although much of the research has focused upon aggressive rather than other antisocial behaviours. Studies in the past decade show that the genetic effects appear to vary according to subtype. Children with callous-unemotional traits show much stronger heritability for antisocial behaviour than children without such traits (0.81 vs 0.30 respectively) [15]. In addition, more aggressive children who offend early have an increased heritability to do so [15].

Other biological risk factors include prenatal or perinatal exposure to toxins and early physical damage to the frontal lobe and other regions of the brain [16]. Research indicates a possible link between serotonin levels in the brain and aggressive behaviours, although the exact nature of this link has not been demonstrated [16].

Young people with aggressive behaviours have been shown to experience general autonomic underarousal, as demonstrated by lower heart rates and skin conductance, indicating an associated lack of inhibitory anxiety, which may protect against antisocial behaviours [16].

Innate temperament, which may be apparent in very early childhood, has been shown to be predictive of future behavioural disorders. Children with vulnerable temperamental characteristics are more likely to be subject to poor parenting styles. Moreover, adoption studies suggest that these

children's behaviour may exacerbate a negative parenting response, leading to an additive effect [17]. Whilst attachment and conduct disorders display similar behavioural manifestations, supporting evidence for an aetiological connection between the two is weak [16].

Although cognitive and reading impairments are often thought to be related to behavioural disorders in children, research evidence is inconsistent due to confounding variables such as ADHD, poor school attainment and gender. Other factors including impulsivity and social withdrawal have been shown to be associated with antisocial behaviours, as have social skills deficits such as failing to notice relevant social cues, whilst misattributing hostile intent to others [16].

Children from socially disadvantaged areas have higher levels of conduct disorders [18]. However, much of this effect is thought to be mediated by intrafamilial social processes associated with poor parenting and parental psychopathology [16], including mental illness, and alcohol and substance misuse.

Certain parenting styles have been consistently shown to link to behavioural disorders in children, including lack of parental involvement, harsh and inconsistent discipline, and poor monitoring and conflict management. Children who have been exposed to sexual or physical abuse have a significantly increased risk of developing CD [16]. Peer relationships and community factors such as drug availability and crime rate may also influence the development of behavioural problems in children [16].

PREVENTION AND TREATMENT

There are many good reasons for trying to alleviate childhood behavioural disorders. As well as causing distress and damage to individual children and families, conduct disorders are known to have a considerable cost to the wider society. Scott *et al*. have shown that by the age of 28 years, costs for people with CD were 10 times higher than for those with no problems. These costs include crime, extra educational provision, foster and residential care, and state benefits as well as smaller costs to the health service [19].

As parenting practices have been identified as relevant aetiological factors in the development and maintenance of childhood behavioural disorders, there has been considerable interest in the use of parenting programmes as a form of prevention and treatment.

In 2006, the National Institute for Health and Clinical Excellence (NICE) and Social Care Institute for Excellence (SCIE) jointly commissioned a review of parenting programmes in the management of children aged 12 years or younger [1]. They concluded that group-based parenting/education programmes are to be recommended in the management of children with conduct disorders. For those parents with whom it is difficult to engage or for whom problems are more complex, similar individual-based programmes can be used instead.

Parenting programmes aim to improve the child's behaviour by helping parents to change the ways in which they approach parenting and to improve their relationship with their children. Whilst most group-based programmes focus upon the actions of parents without the direct involvement of the child, some individual programmes include observation of parent–child interactions, allowing these to be modified as necessary. Most parenting programmes comprise behavioural management techniques based upon social learning theory. These teach parents how to increase desired behaviours through positive reinforcement, whilst decreasing unwanted behaviours by reducing social reinforcement, such as by ignoring behaviours or using Time Out techniques. Programmes will also usually include elements to help parents to understand their children's feelings and behaviours and subsequently improve understanding and communication between parent and child [1].

Analysis of relevant research studies indicates that such interventions are clinically effective at improving children's behaviour, may lead to an improvement in maternal mental health, and are cost-effective ways of treating children with conduct disorders [20].

There is now a wide variety of group-based parenting programmes devised to prevent the development of childhood behavioural disorders as well as to help treat those children who have already developed such problems. Examples of effective programmes include the Triple P, Positive Parenting Programme and the Webster–Stratton Incredible Years Programme, with evidence that these effects continue over several years [18]. Several long-term follow-up studies in the USA

and Canada have shown a reduction in later criminal activity in those children exposed to a variety of early interventions, with huge cost benefits to society [21].

In some circumstances parent training programmes may not be feasible or as effective as hoped. Some families may be unwilling to take part in such programmes or there may be additional risk factors in the child, such as callous and unemotional traits, which may reduce the potential effectiveness of this approach. In these cases, NICE recommends that alternative approaches such as individual cognitive problem-solving skills training should be considered [22]. Other theoretical models such as attachment theory, systems theory or cognitive attribution theory may also be helpful [23].

As children with conduct disorders often present with other comorbid child mental health disorders, these disorders may require treatment in their own right, which may in turn lead to a reduction in the behaviour problems. Studies have suggested that the treatment of ADHD with stimulants or atomoxetine may lead to an improvement in comorbid oppositional behaviour, whilst atypical antipsychotics have been shown to be effective for the treatment of acute and chronic aggression in young people with learning disabilities or pervasive developmental disorders [24].

It is generally agreed within the scientific community that short-term interventions such as military-style boot camps, whilst often promoted within certain sections of the British media, are not effective in the long term [24]. Frightening children with the aim of reducing aggressive behaviour but without offering them any other behavioural alternatives, has the opposite effect of that intended, perhaps as a result of an increased fear-aggression reaction or due to modelling of deviance [24].

CONCLUSION

Childhood behavioural disorders have always been and still remain a common problem and, given current epidemiological trends, are likely to continue to do so for the foreseeable future. These disorders lead to considerable damage, both in terms of the quality of life for young people, their families and their victims, and the wider economic cost to society as a whole. Simple behavioural disorders can progress to much more serious personality disorders in adulthood. We know that there are effective treatments for conduct disorders and these are increasingly being made available across the country via community parenting programmes. Our challenge remains to identify which factors within such programmes may be effective upon different symptom subtypes, to help identify those children who are not responsive to these approaches and consider which alternative methods may be best employed in these cases, and to spread effective treatments to those hard-to-reach families who perhaps need these most.

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29

Specific Language Impairment

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WHAT'S NEW

- Children with specific language impairment (SLI) are late talkers. Language loss in early infancy is not a feature of SLI. Language loss appears to be strongly and specifically associated with autism spectrum disorders (ASD).
- Difficulties with the comprehension of language appear to be prognostic. Lower abilities to understand language are associated with poorer outcomes. Poor language comprehension is a marker of SLI.
- SLI is a relatively stable condition in middle childhood. Group data suggest that, on average, children with SLI do not catch up with their peers nor do they fall further behind. They develop language at a strikingly similar rate to their typically developing peers, maintaining the degree of attainment/impairment they experienced at around 7 years of age.
- Oral language abilities matter throughout development. There is a need for the assessment of oral language beyond childhood in children with SLI. Also, language assessment is crucial in children and adolescents who present with difficulties in learning (mild-moderate learning disabilities), literacy (including dyslexia), behavioural (conduct), emotional and social functioning (including broader phenotype ASD).

WHAT IS SPECIFIC LANGUAGE IMPAIRMENT?

Specific language impairment (SLI) is usually defined as language difficulties in the context of adequate non-verbal skills, normal hearing, absence of frank neurological damage or autism. There is variation in precisely how SLI is diagnosed (see Box 29.1). Nonetheless, there is consensus that SLI represents impairments in language that are disproportionate to difficulties in other non-linguistic domains.

Typically, SLI comes to the attention of clinicians as a result of concern from significant others about the child's progress with language learning. In practice children with this condition rarely have difficulties *only* with language. Furthermore, there is considerable variation not only in the severity but also in the nature of the language difficulties evident in SLI. These considerations raise challenges for the diagnosis, explanation and treatment of the disorder in particular, as SLI presents substantial issues calling for the investment of clinical,

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Box 29.1 Definitions of specific language impairment (SLI)

SLI is generally defined in terms of a discrepancy between non-verbal cognitive functioning and language skills, as measured on standardized tests. Debate centres on the required size of that disparity, the level of language impairment required, and the measurement of verbal and non-verbal abilities. For example:

- The *International Classification of Diseases, 10th revision* (ICD-10) requires language difficulties greater than 2 standard deviations (SD) *below* the mean, with verbal skills at least 1 SD *below* measures of non-verbal cognitive functioning.
- The *Diagnostic and Statistical Manual of Mental Disorders-IV-Text Revision* (DSM-IV-TR), requires *substantially worse* performance on measures of verbal abilities compared to non-verbal cognitive functioning. What constitutes 'substantial' is not defined operationally, though *functional impairment* is required – i.e. SLI interferes with academic or occupational achievement, or with social interaction.
- *The clinical research definition of SLI* [19] works with threshold and discrepancy measures that enable consistent identification of SLI by speech and language therapists. It requires a combination of language difficulties, assessed on a composite standardized language measure, that fall 1.25 SD *below* the mean (approximately, the 10th centile). Plus, adequate non-verbal cognitive functioning (i.e. a Performance IQ *greater* than 1 SD below the mean, equating to a standard score of 85 or higher).

educational and public health resources. SLI is a common disorder, estimated to affect approximately 6% of the population, with boys being more affected than girls at a ratio of 2:1. Although SLI is the term most commonly used in research, it is worth noting that it is rarely used in clinical settings, where the condition is more likely to present under a variety of other names. These can include: 'language disorders' subdivided into 'expressive' and 'mixed expressive-receptive' types [*Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition – Text Revision* (DSM-IV-TR) and proposed DSM-5]; 'speech, language and communication needs' (Royal College of Speech and Language Therapists); 'developmental language delay', 'language impairment', 'primary language difficulties' and other similar names (educational practice settings).

WHAT CAUSES SPECIFIC LANGUAGE IMPAIRMENT?

It has become clear that SLI is not a single-cause disorder. A number of theories have been put forward, all of which have received some empirical support. It seems likely that multiple risk factors are implicated. These include genetic, neurobiological, cognitive and environmental influences.

Biological bases of SLI: genetic and neurobiological factors

There is strong evidence that SLI runs in families. The majority of children with SLI have a positive family history of language difficulties, with a first-degree relative usually affected. The contribution of genetic factors is most clearly indicated in twin studies, where identical twins have a much higher concordance for SLI than non-identical twins [1]. Patterns of inheritance appear to be complex, involving interactions among multiple genes [2]. Technological advances have made it possible to examine brain development in children with SLI. However, few atypicalities have been identified. The most consistent neuroimaging findings suggest leftward asymmetry and a reduction in cerebral volume. Electrophysiological evidence suggests abnormal auditory processing [3]. However, these abnormalities have also been observed in other developmental disorders. Thus, further research is needed to identify distinctive features of brain development in individuals with SLI.

Cognitive bases of SLI: non-linguistic and linguistic factors

Different approaches emphasize different systems as influential in the aetiology of SLI. One prominent approach highlights memory impairments in SLI.

- Phonological short-term memory deficits have been extensively documented, and there is evidence of heritability of this type of difficulty [4]. Such an impairment has a key impact on the child's ability to retain verbal information long enough to develop accurate speech and language representations.
- More general impairments in working memory have been identified in SLI, and more recently difficulties with implicit, procedural memory [5]. Such deficits affect children's ability to extract the regularities of the language they are learning, such as suffixation, as in the use of regular past tense 'ed' in English.

Other views focus on limitations in other areas of cognitive functioning such as perceptual and information processing capabilities, temporal auditory processing capacity and executive functions. Furthermore, some accounts assume that language structures are autonomous of other cognitive systems. These theories emphasize linguistic factors and suggest SLI involves deficits or immaturity in systems responsible for the representation of grammar, of linguistic features, or of structural relationships.

Environmental influences

In general, samples of children with SLI contain disproportionately high numbers of individuals from socioeconomically disadvantaged backgrounds. This could be interpreted as an outcome of disadvantage or as a consequence of intrafamilial transmission, or as due to some more complex interaction. Overall, present evidence supports the assumption of multicausality.

WHAT TYPES OF LANGUAGE DIFFICULTIES DO CHILDREN WITH SLI HAVE?

The heterogeneity of SLI is widely recognized. A number of different classification systems have been devised. The more enduring clinical types differentiate children with only 'expressive' language problems versus children with both 'expressive and receptive' difficulties.

- *Expressive SLI (E-SLI):* Children exhibit mainly language production difficulties in the context of adequate comprehension abilities. Subtle comprehension deficits, however, can be detected when using sufficiently sensitive instruments.
- *Mixed expressive-receptive SLI (ER-SLI):* Children have difficulties with both language comprehension and language production. Difficulties are usually evident at the word (vocabulary) and sentential levels, especially complex sentences.

These two types of SLI are recognized by DSM-IV-TR [6], ICD-10 (*ICD-10 International Classification of Mental and Behavioural Disorders in Children and Adolescents*) [7], and the clinical research definition of SLI. However, it is not clear if these profiles of impairment are qualitatively different or represent different points on a continuum of severity, with ER-SLI representing the more severe cases. More recently, a third clinical type has received attention. This is children in whom the social use of language, that is, pragmatic abilities, is the most prominent difficulty. Vocabulary and grammar can be relatively strong, that is, sentences may appear well formed, but comprehension of extended discourse is usually poor and social interactions can be odd (verbose or overformal with poor turntaking skills). These children are currently referred to as having pragmatic language impairment (PLI). There is debate regarding the overlap between PLI and autism spectrum disorders (ASD).

Major types of SLI in middle childhood

Expressive SLI is thought to be more common in the preschool and early school-age years. By middle childhood, the most common profile of SLI is mixed expressive-receptive SLI. A small proportion of children have PLI (see Figure 29.1 for major types of SLI in middle childhood).

DISTINCTIVE FEATURES OF LANGUAGE IN SLI

The language difficulties of children with SLI can be manifested in various areas of language functioning at varying levels. However, there are some noteworthy areas of difficulty.

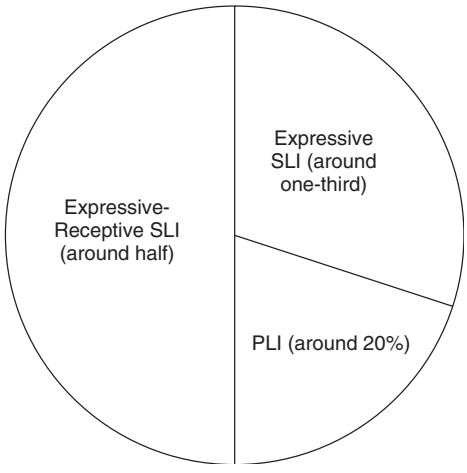

Figure 29.1 Distribution of major types of specific language impairment (SLI) in middle childhood. PLI, pragmatic language impairment.

The majority of children with SLI learning English have difficulties with grammar, in particular verb morphology. For example, they fail to mark tense accurately (they say 'play' for 'played', omitting 'ed') and do not always use auxiliaries ('I staying there' for 'I am staying there'). These difficulties are evident even when children with SLI are compared to younger typical children learning language. Grammar can be disproportionately affected in SLI and has been suggested as a hallmark of the disorder [8].

DEVELOPMENTAL PROGRESSION OF LANGUAGE SKILLS IN SLI

Specific language impairment is characterized by language difficulties from the outset of the language-learning process. Instead of reaching developmental language milestones on schedule (first words between 12 and 24 months, word combinations between 24 and 30 months of age), children with SLI are slow from the beginning. It is a hallmark of SLI that these children are late talkers: they are late in acquiring their first words and in putting together their first word combinations. It is not the case that children with SLI start developing language normally and then stop and become delayed or lose what they have learned. Occurrence of 'language loss' in infancy is reported in some children with autism spectrum disorders (ASD) but *not* in children with SLI. This feature appears to distinguish between the two disorders [9], and can be particularly useful for the differential diagnosis between SLI and ASD in the preschool period. In childhood, difficulties with the sound system of the language, that is, phonology, can co-occur with SLI. However, by middle childhood problems with sound production are usually resolved or less evident (unless there are oral-facial motor difficulties/apraxia), and most children with SLI are intelligible.

It used to be thought that SLI was a short-term difficulty in language learning that, with support, could be resolved by the early school years. Although this is true for a proportion of children experiencing transient language delay (approximately 40%), developmental follow-up studies have shown that children with SLI have persisting language difficulties well into adolescence and even adulthood [10]. Research on the growth trajectories of language-impaired individuals from childhood to adolescence is only just emerging. The evidence suggests that children with SLI show similar, parallel patterns of language growth in comparison with their typically developing peers. The level of language that children with SLI reach at 7 years in relation to their peers is predictive of their level of language attainment in middle childhood and adolescence. As a group, individuals with SLI do not 'catch up' with their peers nor do they fall further behind. Children with SLI develop language at a strikingly similar rate to their typically developing peers, maintaining the degree of attainment/impairment they experienced in childhood (Conti-Ramsden *et al*., unpublished).

ASSOCIATED DEVELOPMENTAL PROBLEMS AND OUTCOMES

Language is fundamental to human behaviour. Hence, it is not surprising to find that individuals with language impairments tend to have associated difficulties in other aspects of their lives. For example, recent research indicates developmental interactions between oral language skills and areas of functioning such as literacy, memory skills and more general non-verbal abilities throughout middle childhood, adolescence and beyond [11,12].

Still more broadly, there is evidence that young people growing up with SLI experience greater

difficulties in social interaction than do typical children and adolescents [13]. These children are more vulnerable to social exclusion, behavioural and emotional difficulties, and to being bullied [14]. Their linguistic and literacy problems impact on their educational progress and attainment and on their uses of new technologies [15]. As adolescents, they have greater difficulties in dealing with the myriad tasks of autonomous daily life [16,17]. A proportion of children with SLI develop broader phenotype autistic symptomatology in adolescence [18].

There is much debate with regard to the processes underpinning the above-mentioned developmental observations: Do they reflect the impact on language development of still more fundamental cognitive and perceptual capacities? Are they manifestations of comorbid conditions that emerge during the developmental process? Are they developmental manifestations of bidirectional interactions between language impairment and other areas of functioning? The answers to these questions require further empirical research and clinical data. However, what is clear is: (i) that oral language abilities matter throughout development; (ii) that children with SLI are likely to demonstrate a range of associated difficulties; and (iii) that these children are at risk of less successful developmental and educational outcomes.

IMPLICATIONS

There is a need for the assessment of oral language abilities in children with SLI in middle childhood and beyond. Furthermore, because of the observed changing profiles of these individuals, when children and adolescents present with difficulties in learning (mild-moderate learning disabilities), literacy (including dyslexia), behaviour, or emotional and social functioning (including broader phenotype ASD) they should be assessed for their oral language skills. This is important not only because they may be in need of support for their development but because many of the foundations of intervention in clinical, educational and mental health practice involve the oral verbal medium. For example, language is the mode of educational instruction and a key element in interactions between clinician and client in cognitive–behaviour therapy.

All practitioners who work with young people with developmental difficulties need to be sensitive to the possibility that these children may have, among other problems, language impairments. When we have a young person walking through our door, we need to be more aware of the potential need to evaluate and, if required, obtain support for his or her oral language skills. Only in this way will we be able to make it possible for individuals with SLI to have a good quality of life, find employment, and establish long-term relationships.

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Section 5c

Adolescence

30

Depression and Suicidal Behaviour in Children and Adolescents

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This chapter focuses on depressive disorder – as defined by ICD-10 (*ICD-10 International Classification of Mental and Behavioural Disorders in Children and Adolescents*) and DSM-IV-TR (*Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition – Text Revision*) – and suicidal behaviour. Whilst suicidal behaviour may be indicative of a range of difficulties, it may also be a symptom of depressive disorder. The epidemiology, aetiological factors, course, diagnostic assessment and management for depressive disorder and suicidal behaviour will be described.

DEPRESSIVE DISORDER

Whilst there was little recognition of depressive disorders in children and adolescents before the 1970s, the use of symptom-oriented psychiatric interviews with children and adolescents led to an acknowledgement that depressive disorders resembling those seen in adults do occur in this age group; the current diagnostic criteria are the same as those used in adults – mood change (low mood/irritability) or loss of enjoyment lasting at least 2 weeks, associated cognitive (e.g. guilt, pessimism about the future, suicidal ideation) and biological (e.g. change in appetite, sleep disturbance, fatigue) symptoms, together with functional impairment (at home, school or with regard to peer relationships).

Epidemiology

The prevalence of depressive disorder increases from childhood to adolescence, with a reported prevalence in community samples of adolescents ranging from 1% to 8% [1–3]. It is equally common in girls and boys during childhood, but during adolescence the female:male ratio increases to about 2:1 [4]. It has been suggested that pubertal status rather than age is associated with the increase in depressive disorders among adolescent girls [5]. Whilst the prevalence of depressive disorders in this age group has not changed greatly over the last 30 years, recognition and treatment have increased [6]. Psychiatric comorbidity, especially with anxiety disorders, conduct disorder and substance misuse, are common [7].

Aetiological factors

The aetiology of depressive disorder is multifactorial [8]; risk factors may be divided into those that predispose to (increase vulnerability to) and precipitate (lead to its development at a specific point in time) a depressive episode. These influences act through biochemical and psychological processes. Once established, depressive episodes may be prolonged by maintaining factors that treatment approaches aim to alleviate. These risks are described more fully in Table 30.1.

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Table 30.1 Aetiological factors for depressive disorders.

Risk factor	Evidence
Predisposing factors	
Genetic factors	Greater genetic influence for adolescent than for childhood depression
	Children of depressed parents at greater risk
	Twin studies — heritability 15—80% for depressive symptoms
	Indirect genetic influences, e.g. increased risk of experiencing more negative life events
Family environment	Low levels of parental warmth, high levels of hostility and conflict are associated with increased depressive symptoms
	Parental mental health problems impact on parenting, making it more difficult to meet the child's emotional needs and provide a confiding relationship
Temperament/personality	Children who are slow to adapt to new experiences, socially reticent, easily upset
	Elevated levels of anxiety, high self-criticism and negative attributional style — tendency to blame self rather than others
Early/chronic adversity	Poverty/social disadvantage
	Physical,sexual or emotional abuse
Neurobiological factors	Underactivity of cerebral amine systems
	Abnormalities in cortisol secretion
	Functional and anatomical brain differences in depressed and non-depressed young people
Precipitating factors	
Stressful life events	Examples include losses (e.g. parental separation or bereavement), disappointments and failures (e.g. peer problems, bullying, academic difficulties, failing exams)
Maintaining factors	
Persistent depressive symptoms	Recognized as a risk factor for further depressive episodes
Psychosocial scars	Individuals may experience residual effects from a depressive episode — 'psychosocial scarring', which increases the likelihood of further episodes
Persistent biological/cognitive vulnerabilities	As above
Persistent adversity	Examples include family dysfunction, lack of a confiding relationship with mother, poor peer relationships

Diagnostic assessment

This is facilitated by a mental state examination of the young person via an interview with him/her alone; adolescents themselves are the most accurate informants about internalizing symptoms, which parents may not be aware of. Depressive disorder is often associated with psychiatric comorbidity (40–70%) [4], particularly dysthymic disorder, anxiety disorders, eating psychopathology, conduct disorders and substance abuse. It is important to recognize comorbidity as this has implications for management and outcome.

Outcome

The outcome of depressive disorder (assessed by episode duration or risk of recurrence) differs according to the population studied (mental health service referred or community); it is influenced by factors including age, symptom severity, past history of depressive episodes, comorbid psychopathology and family factors, for example, conflict and parental psychopathology. Recovery is the norm, with 88% recovering within 1 year in community samples [9], and 80–90% by 12–18 months in clinic samples [10,11]. The median duration of depressive episodes is 9 months in clinic-referred samples [12] and 8–12 weeks in community samples [13] – the former generally having more severe episodes. Recurrence is frequent: 12% relapse within 1 year in community samples [13], and 27% within 9 months for clinic samples [14]. Continuity into adulthood is high, with an increased risk of self-harm, completed suicide and impaired psychosocial functioning [15].

Management

The aims of management are:

1. to make an adequate assessment;
2. to treat the depressive disorder, and reduce associated psychosocial impairment;
3. to manage associated comorbidity and risk factors;
4. to prevent relapse.

Initial assessment: This largely depends on the context in which the young person is seen and the expected level of severity of the problems. Thus, in primary care settings where youngsters with milder depression are seen, the brief assessment will focus on mood, including self-harm risk, and current difficulties including social function. Those seen in specialist child and adolescent mental health services are likely to have more severe depression with more comorbidity and complex family situations. In this context a more detailed assessment will cover developmental history and functioning at school, as well as family relationships and other problems.

Treatment: Treatment of brief or minor depression will include exploration of difficulties, activity scheduling, and follow-up. Mild to moderate depression, where social function might be impaired, should be managed initially with psychological treatment [16]. Most frequently used is cognitive–behavioural therapy (CBT), which starts with psycho-education and includes self-monitoring, for example, diary keeping, increasing competence in emotion recognition, challenging cognitive distortions, and activity scheduling. An alternative appropriate psychological therapy is interpersonal psychotherapy for adolescents (IPT-A), which addresses problem relationship areas such as role conflict, transitions or losses. While both CBT and IPT-A have evidence for effectiveness [16] there are currently few child mental health professionals in the UK trained in IPT-A, but CBT is becoming widely available.

More persistent moderate or severe depression will require antidepressant medication. Recent studies, predominantly with adolescents, suggest that selective serotonin reuptake inhibitors (SSRIs), particularly fluoxetine, are helpful [17]. In recent years there has been a high level of concern regarding the possible increase of suicidal events with the use of SSRIs. Although the increased risk is slight, close monitoring is appropriate. Failure to respond to fluoxetine can be managed with a change to another SSRI, or another class of antidepressant such as venlafaxine, with the addition of CBT [18]. Poor progress or high risk of self-harm may require psychiatric admission.

Managing associated comorbidity and risk factors: The presence of comorbidities and associated risk factors means that additional interventions may be required. The associated anxiety or conduct problems might require specific interventions. For some youngsters if the associated disorders are effectively treated the depression might lift. Addressing

problems in family relationships, in school or with peers will require specific interventions.

Preventing relapse: If medication achieves remission it should be continued for 6–9 months. Psychological treatment sessions may also be required after the depression has improved. While there is little evidence about the best way to prevent relapse it is likely that recognition of stressors, early identification of symptoms and early referral to specialist services is appropriate. Options will be booster sessions of CBT or a short course of antidepressants.

SUICIDAL BEHAVIOUR

Epidemiology

Suicide is very uncommon in childhood and early adolescence but the rate increases markedly in mid-adolescence. World Health Organization (WHO) data from 2004 indicate that the UK suicide rate for males aged 15–24 was 8 per 100,000 as compared with 2.3 per 100,000 for females. Males tend to use more violent methods, and rates vary by country and ethnicity.

Deliberate self-harm (DSH) is common in adolescents; studies report a 12-month prevalence rate of 7–9% [19,20], and it is approximately three times more common in females. However, only a minority (12.6%) of DSH episodes lead to hospital presentation [20,21]. The most common methods are self-poisoning and cutting. The term DSH is frequently used as it does not imply a specific level of suicidal intent.

Thoughts of suicide (in the absence of deliberate self-harm) are not uncommon (approximately 15% in the previous year), and are more frequent in females [20].

Aetiological factors

These may be divided into predisposing factors (e.g. within the young person, their family and the wider environment) and precipitating factors.

Predisposing factors:

- *Individual:* Psychiatric disorder, especially major depressive disorder, but also anxiety, substance misuse and conduct disorder, are key risk factors for DSH [20]. In the context of depression, feelings of hopelessness, despair, low self-esteem and a tendency to self-blame are particularly relevant. Psychological factors such as impulsivity and poor problem-solving skills reduce the ability to discuss and contemplate difficulties [22], and in this context DSH may represent an impulsive response to problems in an attempt to find an immediate relief for distress or an escape from a troubling situation, rather than using problem-solving strategies or accessing social support to work out a solution. Young people who are socially or emotionally isolated, and particularly those who lack a family confidant(e) with whom they can share problems, are at increased risk of self-harm [23]. Young people who have experienced abuse, particularly physical and sexual abuse, are at greater risk of DSH [20,21,24]. A history of DSH is predictive of future episodes; up to 30% report a previous episode (which may not have come to medical attention) [25].
- *Family:* Communication difficulties within the families of young people who self-harm are typical; adolescents who self-harm (compared to those who do not) are less likely to feel able to talk to their parents. This is also a risk factor for repeated compared to a single episode of self-harm [26]. A family history of mental health problems, particularly parental DSH, is an additional vulnerability factor. Parental divorce is also more common in families of young people who self-harm [20].
- *Wider environment:* School problems may be very relevant in this age group and include academic difficulties leading to underachievement and pressure to achieve, as well as bullying. Difficulties with regard to relationships with peers, boy/girlfriends and teachers are also aetiologically important. Exposure to suicide or suicide attempts in family or friends also increases risk [25].

Precipitating factors: Deliberate self-harm is frequently precipitated by stressful life problems; often these are interpersonal conflicts or difficulties with parents or siblings, such as arguments, or rejection by boy/girlfriends or peers, and school problems such as academic difficulty and bullying. It is frequently an impulsive act, with many individuals thinking about it for just minutes before acting. Over 50% consult their GP in the month before

deliberate self-harm but presentation is generally not with psychological symptoms [27].

Risk associated with self-harm

The factors associated with high risk from self-harm are given in Box 30.1. The physical severity of the self-harm is not a good indicator of intent as young people are often unaware of the objective degree of lethality of specific substances and quantities; it is their belief about potential lethality that is important.

Box 30.1 Factors associated with high suicidal intent

- Carried out in isolation
- Timed so that intervention is unlikely, e.g. after parents are at work
- Precautions taken to avoid discovery
- Preparations made in anticipation of death, e.g. leaving directions as to how possessions should be distributed
- Other people informed of individual's intention beforehand
- Advance planning of attempt
- Suicide note
- Failure to alert others following the attempt

Course

At least 10% of adolescents who self-harm do so again in the following year; this is especially likely in the first two or three months. Factors that increase the likelihood of repetition include previous self-harm, personality disturbance, depression, substance misuse, extensive family psychopathology, poor social adjustment, social isolation and a poor school record [25]. Approximately 0.5% eventually kill themselves; risk factors include male gender, older age, high suicidal intent, mood disorder, substance abuse, violent method of self-harm and previous psychiatric admission.

Management

The aims of management are:

1. to make an adequate assessment;
2. to treat the depressive disorder, and reduce associated psychosocial impairment;
3. to manage associated psychiatric disorder and risk factors;
4. to prevent further episodes of DSH.

Type of assessment: This will depend on the context in which the young person is seen [28]. Thus, in primary care settings the main goal is to ascertain risk and consider whether self-harm has actually taken place, as this will often require referral to the appropriate local hospital accident and emergency service. In the hospital setting paediatric management is required for physical effects of self-harm, coordinated with child and adolescent mental health assessment, and social work input. When the young person is referred to the out-of-hours hospital accident and emergency service, existing guidance is that admission is required overnight with the assessment taking place the following day [28]. The mental health assessment requires the identification of a psychiatric disorder and the range of risk factors. The assessment should include interviewing the young person alone as well as with his or her parent(s). The purpose of this assessment is: (i) to assess the current risk with regard to suicidality and further deliberate self-harm; (ii) to understand the young person's and the family's difficulties and how these have led to self-harm; (iii) to determine whether the young person is suffering with a psychiatric disorder, for example depression (and the level of hopelessness), or drug or alcohol misuse; and (iv) to assess the resources of the young person and the family. It is important to establish whether the index episode of deliberate self-harm was associated with a high degree of suicidal intent (see Box 30.1); whilst a minority of patients may try to conceal their true intent, assessment of intent is best facilitated by obtaining a detailed understanding of the circumstances of the attempt and comparing this information with factors known to be associated with high intent. The outcome of this assessment will inform discharge and further management planning.

Treatment: This requires that the young person should be kept safe, which means restricting access to potentially harmful substances, such as drugs, used for self-harm, as well as alcohol. Appropriate care and emotional support are needed [29].

This often requires family intervention, and two main approaches have been described. Family-based problem-solving therapy aims to improve communication and reduce conflict in the family. This may be effective for adolescents who are not depressed [30]. Family systems-oriented therapy will address problems in family organization, communication and affect. A newer intervention is dialectical behaviour therapy, which aims to improve self-acceptance, increase assertiveness and reduce interpersonal conflicts, and avoid situations that trigger distress. Cognitive–behavioural therapy for DSH has also been described and has a clear rationale [31]. Drug treatments have not been shown to be effective, although may have a role in the treatment of underlying psychiatric disorder. Overall, the evidence base for treatments following DSH is weak. Unfortunately less than one-half of adolescents who self-harm, and their parents, will remain in therapy after the initial assessment. However, the assessment will reveal specific psychiatric disorders, such as depression, in a significant proportion of cases and treatment should then be targeted at the underlying disorder.

Prevention: The main elements are identification of those at highest risk by the prompt recognition of depression or other problems associated with suicidal behaviour; establishing crisis intervention; and reducing access to methods of self-harm, such as decreasing the availability of poisonous domestic gas, and restricting the pack size of analgesics in the UK [29].

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31

Eating Disorders in Adolescence

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DIAGNOSIS AND CLASSIFICATION

The term 'eating disorder' is restricted to disorders of eating behaviour driven by overvalued ideas about weight and shape. Within this narrow definition, there are two well-described disorders, anorexia nervosa (AN) and bulimia nervosa (BN). AN is characterized by determined food avoidance in pursuit of thinness, resulting in clinically significant weight loss, which may or may not be enhanced by so-called 'compensatory behaviours' designed to counteract the fattening effect of food. The DSM-IV-TR (*Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition – Text Revision*) [1] recognizes a restrictive (AN-R; food restriction and exercise only) and a binge-purging (AN-BP) subtype of AN. Two main features distinguish AN from BN. The first is the centrality of binge eating to BN, characterized by loss of control over eating. The second is that, although in BN thinness is pursued and desired, sufferers are by definition within the normal weight range. DSM-IV recognizes purging (BN-P) and non-purging (BN-NP) subtypes of BN.

Despite features in common, each disorder has a distinct course, outcome and treatment response, with accumulating evidence for differential familial (including genetic), personality, and neurodevelopmental risk. The current challenge, given the overlap in clinical features, is accurately to predict the course and prognosis for a given individual at the time of presentation.

The other important diagnostic issue is that the majority (around 60%) of patients at all ages presenting with a clinically significant eating disorder do not meet full diagnostic criteria for either AN or BN, and would be diagnosed with Eating Disorders Not Otherwise Specified (EDNOS) in the DSM-IV [1], or Atypical AN or BN in ICD-10 (ICD-10 International Classification of Mental and Behavioural Disorders in Children and Adolescents) [2], or be unclassifiable. Common examples of EDNOS include patients with AN-like illness who have lost considerable weight but are still in the healthy weight range or have not lost menses; patients who binge and purge but at a lower frequency than the BN criteria specify; patients who purge but do not binge (purging disorder), or binge but do not purge [Binge Eating Disorder (BED)]; or patients for whom disordered eating is one of a number of risk behaviours or comorbidities [3]. Of more uncertain nosological status are patients who have determined food avoidance that does not appear to be driven by a drive for thinness or fear of weight gain (non-fat phobic AN) [3]. Such presentations are common in young patients, when it is sometimes known as Food Avoidance Emotional Disorder [4], and in non-Western cultures and minority ethnic groups. It is likely that BED will be included in the DSM revision, while other presentations such as 'purging disorder' [5] and 'non-fat phobic AN' require further research.

Developmental issues with respect to diagnosis include the degree of reliance on self-reported cognitions, developmental differences in the impact on physical health, and the way that parental reporting of behaviours and eating concerns is assimilated into the diagnostic process [6]. The diagnostic

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process should include a family interview, a medical assessment, and an individual assessment with the young person. Core eating disorder cognitions are best identified using a semi-structured diagnostic interview such as the Eating Disorders Examination (EDE) [6,7]. Key diagnostic questions include asking how much the young person would like to weigh, how they feel about their weight and shape, and whether they or anyone else is worried about their eating or exercising.

EPIDEMIOLOGY AND AETIOLOGY

Some form of eating disorder is experienced by 3–12% of adolescents [8,9]; most would be diagnosed with EDNOS. For many this will be a transient period of eating pathology, with recovery rates at 1 year of around 91–96% [9]. In an adolescent population, the prevalence of full syndrome AN is around 0.3% (range 0–0.9%) in 11–15-year-olds [10], but because of its chronicity once established, AN is often cited as the third commonest chronic illness of adolescence. For BN the average prevalence is 1%, but of these only around 5% will reach mental health services [10]. The hidden nature of eating disorders means that when patients do present, often as a result of parental concern, the illness is often well established, and should therefore be taken seriously from the first consultation [11].

Eating disorders are biopsychosocial disorders of complex aetiology; no single factor is sufficient to account for onset or maintenance of any given presentation. Table 31.1 outlines the best established risk factors, as well as common behavioural indicators of a potential eating disorder, suggesting that a full assessment is indicated. Familial factors are important; female relatives of someone with a clinical eating disorder is more than four times as likely to have BN and more than 11 times as likely to have AN than someone with no family history of eating disorders. This figure is probably higher for subclinical or partial syndromes. From twin studies, AN has an estimated heritability of 58–76% and BN of 31–83% [8]. There is emerging evidence that specific cognitive profiles in terms of cognitive inflexibility, cognitive inhibition, visuospatial construction and memory, may be relevant to the aetiology of AN [9], and neuroimaging studies show persistent processing deficits in limbic function [12]. There is also increasing recognition of impaired 'social cognition' in a proportion of young people with AN [13], which may have implications for treatment style and treatment response.

A formulation of individual, systemic and cultural factors, divided into predisposing, precipitating, perpetuating and protective factors, is helpful in teasing out the elements important for any one individual patient, and can be a therapeutic tool to aid engagement. An example is given in Table 31.2. In eating disorders there is an interplay between dietary restraint, weight and eating, with issues such as negative affect, low self-esteem, adversity, shame, feelings of personal ineffectiveness or powerlessness, and for young people specifically, issues around growing up, identity formation/finding a voice, learning about risk taking and risk avoidance, other people's issues, and cultural pressures. The formulation gives a starting point for disentangling these themes.

Table 31.1 Risk factors for and behavioural indicators of eating disorders.

Risk factors for developing an eating disorder in adolescence	Psychological or behavioural markers of an eating disorder
Female sex	Reluctant attender
Repeated dieting	Seeks help for physical symptoms
Early puberty	Resists weighing and examination
Temperament – perfectionist personality	Covers or hides body with loose clothes
Teasing about weight and dieting	Secretive/evasive
Low self-esteem	Increased energy ± agitation
Losses and major life events	Gets angry when confronted
Family history of eating disorder	

Table 31.2 Hypothetical example of a formulation for an adolescent who has developed an eating disorder.

	Individual	Systemic	Cultural
Predisposing	Perfectionist nature Picky eater from a young age	Grandmother hospitalized for weight loss as a teenager	
Precipitating	Onset of menses Falling out with best friend	Older sister dieting	
Perpetuating	Social avoidance Low mood		Highly competitive group of friends
Protective	Enjoys school	Intact, motivated and supportive family	Supportive school Maintained some links with peer group

Table 31.3 Indicators of high risk.

Indicator	Comment
Very low weight or rapid weight loss	Less than 70% of BMI for age and gender or loss of over 1 kg for consecutive weeks in a low-weight child
Bradycardia	Symptomatic or with asymptomatic awake and resting heart rate $<$45 bpm
Postural hypotension	Symptomatic or asymptomatic with a postural drop in systolic blood pressure of greater than 15 mmHg (note some authorities recommend admission if drop greater than 10 mmHg)
Severe electrolyte imbalance	E.g. potassium $<$3 mmol/L, hyponatraemia or hypernatraemia Hypoglycaemia
Severe-to-moderate dehydration	Difficult to assess clinically; will rely on history too
Other severe medical complications	E.g. seizures or pancreatitis, hypothermia
Psychiatric reasons	E.g. suicidality, self-harm (e.g. head banging) or aggression
Child protection reasons	Violence from sufferer towards others, or towards the sufferer; risk of sexual abuse; parent/carer treatment non-attendance

MANAGING EATING DISORDERS

Assessment and management of a young person with an identified eating disorder must tackle medical, nutritional and psychological aspects of care, and be delivered by health-care staff who are knowledgeable about normal adolescent development. When management is shared between primary and secondary care, or between paediatric and mental health services, clear agreement is needed about who is responsible for monitoring patients, and this should be communicated to the patient and his or her family. Consideration should be given to the impact of the problem on siblings, who should be involved in treatment when possible. Admission to hospital is necessary if there is acute physical compromise, high psychiatric risk, or for a specific intensive treatment. Indicators of high risk are given in Table 31.3.

Box 31.1 Medical complications of eating disorders

Medical complications of calorie restriction

- Cardiovascular: ECG abnormalities – bradycardia; T-wave inversion; ST segment depression; prolonged Q-T interval; dysrhythmias (SVT, VT); pericardial infusions
- Gastrointestinal system: delayed gastric emptying; slowed GI motility; constipation; bloating; fullness; hypercholesterolaemia; abnormal liver function (carotenaemia)
- Renal: increased blood urea (from dehydration and reduced GFR) with increased risk of renal stones; polyuria (from abnormal ADH secretion); depletion of Na and K stores; peripheral oedema with refeeding due to increased renal sensitivity to aldosterone
- Haematology: leucopenia; anaemia; iron deficiency; thrombocytopenia
- Endocrine: sick thyroid syndrome (low T_3); amenorrhoea; growth failure; osteopenia
- Neurological: cortical atrophy; seizures
- Death

Medical complications of purging

- Fluid and electrolyte imbalance: low K; low Na; low Cl
- Chronic vomiting: oesophagitis; dental erosions; oesphageal tears; rarely rupture and pneumonia
- Use of ipecac/laxatives: myocardial damage; renal stones; low Ca; low Mg; low KCO_3
- Amenorrhoea

Assessment

The purpose of assessment is to clarify the diagnosis, undertake a risk assessment, assess the impact of the problem on the young person's development and general functioning and the functioning of the family, consider treatment expectations and motivation, and observe family relationships and communication, to reach an understanding (formulation) of the problem with the young person and their family. Assessment also serves to engage the young person and their family, whose motivations for seeking help may be very different. Many young people are brought to treatment, and the egosyntonic nature of eating disorders is such that consent (or assent) to treatment cannot be assumed, but needs to be balanced against acting in the best interests of the child, and the responsibilities, rights and duties of parents to provide, in a manner consistent with the evolving capacities of the child, appropriate direction and guidance. If necessary, formal legal frameworks surrounding child welfare or mental health may need to be invoked, but a collaborative and motivational stance is likely to minimize the need for this except in rare situations.

Medical aspects

Medical complications of eating disorders can be a result of calorie restriction leading to weight loss, poor nutrition or purging behaviours [14]. Box 31.1 summarizes the complications of eating disorders, some of which are short term and some long term. Figure 31.1 shows why body mass index is inappropriate in children and adolescents. In adolescents, degree of underweight is best expressed as percent BMI/median BMI for age and gender (also known as weight for height). Using this terminology, less than 85% BMI would be considered underweight, and less than 70% BMI would indicate severe malnutrition. Weight alone is not adequate to assess medical risk, however. Table 31.3 outlines the risk parameters that require assessment, and when to be concerned. Acute malnutrition is a medical emergency.

In adolescents, assessment of pubertal development is important for determining risk for complications such as growth retardation and

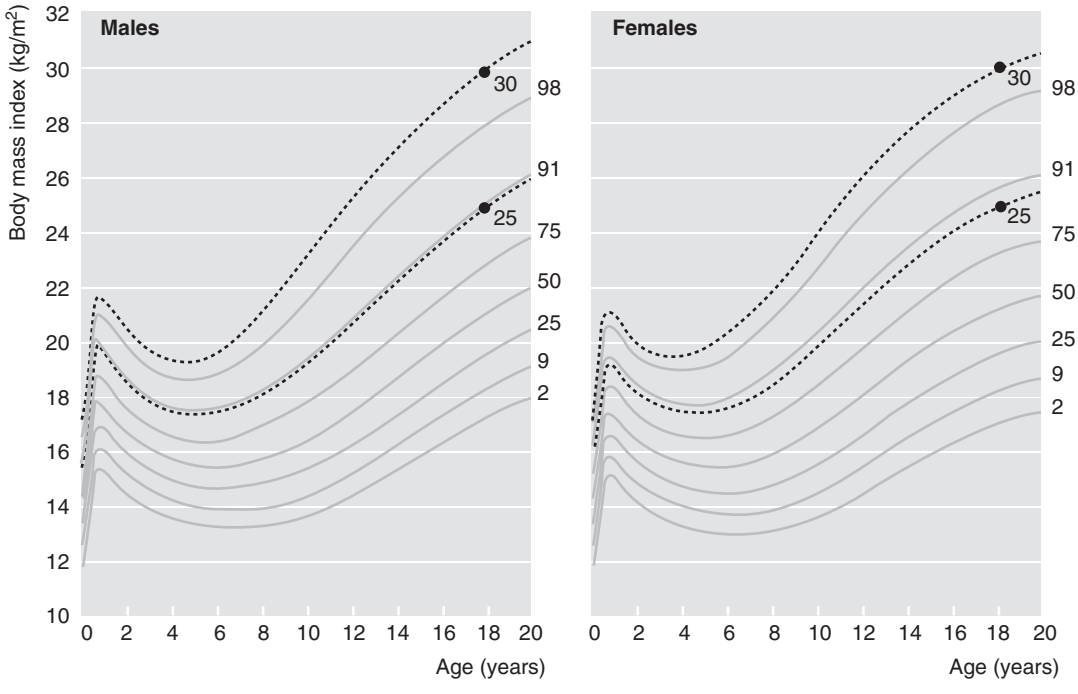

Figure 31.1 Body Mass Index (BMI: weight in kg/square of height in m) varies with age and gender, so centile charts are needed to assess degree of underweight. The red line crosses through BMI of 17.5, defined as underweight in an adult, but which is in the normal range for an adolescent under 16.

osteopenia, and also gives an indication of whether resumption of menses is likely to be the indicator that a 'healthy weight' has been achieved; a patient in early puberty would not be expected to menstruate. Serial pelvic ultrasound can be used to monitor pelvic organ maturation and predict onset of menses [15]. Because puberty in boys runs about 2 years later than in girls, boys are more vulnerable to the impact of low weight on growth and development.

Bones are at risk in eating disorders as a result of endocrine as well as nutritional inadequacy, and adolescence is the time of greatest bone acquisition. The most effective treatment for and prevention of osteopenia is weight restoration and resumption of endocrine function. There is no evidence for the role of calcium or other vitamin supplements, although some guidelines recommend them.

Management of nutritional disturbances in adolescents with eating disorders should take into account the pubertal development and activity level. This is likely to mean that they will need a higher calorie intake for adequate weight gain than the intake required by adult patients with eating disorders.

Psychiatric aspects of management

Eating disorders generate a lot of anxiety, often appropriately, and parents and young people appreciate their concerns being taken seriously, and knowing that the professionals are confident and knowledgeable about the problems. Many seriously ill patients can be managed as outpatients provided an adequately skilled multidisciplinary team is involved and risks can be managed. Involving families in treatment and involving young people in decision-making increases cooperation, motivation and outcome.

Family interventions that directly address the eating disorder should be offered to adolescents with AN [16], usually in the form of

family-based treatment [17], in a conjoint or separated family therapy format [18]. Individual therapy becomes the mainstay of treatment for AN when the young person is ready developmentally to take responsibility for managing their eating disorder, or when the eating disorder has become chronic [19]. This may be in the form of cognitive–behaviour therapy, cognitive analytic therapy or other form of psychotherapy. Treatment should address the eating disorder, including weight and nutritional aspects. Potentially useful psychotropic medications in AN include selective serotonin reuptake inhibitors (SSRIs) for comorbid obsessive-compulsive disorder or depression that has not improved with weight restoration, or atypical antipsychotics such as olanzapine or risperidone [20].

Adolescents with BN can be treated with cognitive–behaviour therapy (CBT) specific to the disorder, with the family included as appropriate [21], or with family-based treatment [22,23]. CBT can be delivered in a CD-ROM format with therapist support [24]. SSRIs are also potentially helpful as an adjunct to psychological treatment for BN.

Inpatient treatment has long been used as a therapeutic option when risks are high or when outpatient treatment response has been poor. Recent studies have questioned the efficacy of inpatient treatment in addressing AN in particular, and even suggested in some cases it may be counterproductive [25]. This has led to efforts to seek alternative treatments for the sickest patients, including increased use of paediatric wards for medical stabilization prior to outpatient treatment, a practice common in the USA [21] and Australia, or intensive family-based outpatient treatments such as multifamily therapy [22].

KEY MESSAGES AND FUTURE DIRECTIONS

Eating disorders are serious mental illnesses [23] with a high morbidity and mortality, but the prognosis is good if appropriate treatment is started early [26], the majority of sufferers recovering within 5 years [27]. Families should be involved in treatment as the main source of support for the sufferer [28]. The trend is away from hospital-based treatment for adolescents, towards treatment that allow relationships with peers and family to be maintained, and for functional aspects of the sufferer's life, such as involvement in education, to be maintained.

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32

Substance Misuse in Young People

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INTRODUCTION

Substance misuse is a major public health problem, with substantial levels of morbidity and mortality. Most children in their middle childhood are exposed to various substances including alcohol and tobacco, and a substantial minority, as high as 10%, continue to use drugs into adolescence and adulthood [1,2]. Many youngsters who misuse drugs have multiple antecedent and coexisting mental health problems, unrecognized learning difficulties, family difficulties, involvement with the justice system and deeply entrenched social problems. Substance misuse takes a high toll in terms of health-care costs, violent crimes, accidents, suicides, social and interpersonal difficulties, and educational impairment [3].

EPIDEMIOLOGY

Estimates from the 2009/10 British Crime Survey suggest that 40% of those aged 16–24 have used one or more illicit drugs at some point in their life, with up to 12% having used illicit drugs in the last month [4]. Tobacco, alcohol and cannabis are the most commonly abused substances, with cocaine and heroin accounting for less than 10% [4,5]. Volatile substance use peaks in early adolescence: about 4–7% of 11–15-year-olds sniffed volatile substances in the last year, and roughly 1% inhale solvents regularly, with the prevalence being substantially higher for youngsters from deprived backgrounds [3] (Table 32.1).

Most of the campaigns against substance misuse are directed at illegal drugs such as cannabis, heroin, cocaine and ecstasy. However, many more people die or develop problems, either directly or indirectly, as a result of using tobacco and alcohol than all illegal drugs combined, and some of the leading experts in the field of addictions have proposed alternatives to the contentious British system of classification of drugs [6].

DEFINING SUBSTANCE MISUSE IN THE YOUNG: A DEVELOPMENTAL PERSPECTIVE

The effects of a drug are not just dependent on the drug itself. The mindset of the individual who takes it and the setting in which it is used are crucial variables. Young people report that they take drugs for a variety of reasons: for pleasure; to conform to attitudes and values of their peer group; to block out traumatic and painful memories; and to relieve sadness and worries associated with their everyday lives. For some young people, the use of drugs and alcohol may become a problem in itself, and a very small minority develop substance dependence. Early onset of substance use and a rapid progression through the stages of substance use are among the risk factors for the development of substance misuse [3]. Longitudinal studies have shown that the highest peak of drug and alcohol use is between the ages of 14 and 18 years, and that most youngsters reduce or stop use by the age of 24 years [7]. The Christchurch Health

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Table 32.1 Use of drugs and alcohol in young people in the UK, 2007/2010.

Substance	Use last year: 16—24-year-olds	Use last month: 16—24-year-olds	Lifetime use/use last year: 11—15-year-olds	Regular use in 11—15-year-olds and 15—16-year-olds
Tobacco	NA	NA	29% (lifetime)	6% (more than one cigarette per day in 11—15-year-olds): 7% in boys and 4% in girls
Alcohol	NA	NA	51% (lifetime)	52% of boys and 55% of girls aged 15—16 engaged in episodic heavy drinking in the last month
Cannabis	16.1%	16%	8.9% (last year)	9% of 15—16-year-olds used in the last month [6] 2% of 15—16-year-olds reported harmful use of cannabis
Cocaine (cocaine powder and crack)	5.6%	2.6%	1.8% (last year)	NA
Ecstasy	4.3%	1.9%	1.2% (last year)	NA
Alkyl nitrites ('poppers')	3.2%	0.8%	1.8% (last year)	NA
Amphetamines	2.4%	0.7%	0.8% (last year)	NA
Opiates	0.3%	0.2%	0.7% (last year)	NA
Hallucinogens	1.5%	0.4%	2.2% (last year)	NA
Volatile substances (glue)	3%	0.1%	5.5% (last year)	NA
Ketamine	1.7%	0.9%	0.6% (last year)	NA

Sources: Reproduced with permission from Flatley *et al*. [4], Fuller, Sanchez [5], and Hibell *et al*. [6].

and Development study estimated that 10% of cannabis users would become dependent, and at the age of 18 years, about 6% were dependent on drugs or alcohol [2].

Given the natural history of substance use in young people and the heterogeneity of the patterns of use, most researchers and clinicians struggle to define what constitutes substance misuse in young people.

Definitions

International classificatory systems – the *International Classification of Diseases, 10th revision* (ICD-10) and the *Diagnostic and Statistical Manual of Mental Disorders, 4th edition* (DSM-IV) – suggest that adult categories like 'harmful use' and 'dependence' (ICD-10) and 'substance abuse' and 'dependence' (DSM-IV) could be reliably used to diagnose substance misuse in

young people. Unfortunately, both systems lack a developmental perspective in psychopathology, and the categories such as 'harmful use', 'dependence' and 'substance abuse' do not seem to capture all stages of substance use in young people [8,9]. For example, tolerance and withdrawal, which typically develop in response to long periods of chronic substance use, are rarely seen in young people.

Alternative classifications in young people

Clinicians and researchers have proposed alternative criteria to classify substance misuse in young people [8,9]. Based on the seminal work by Joseph Novinsky and colleagues, Mirza and Mirza proposed a developmentally sensitive and dimensional model to classify the stage of substance use in young people [10], starting with non-use at one end, moving through an experimental stage, social stage, at-risk (prodromal) stage, and stage of harmful use to substance dependence at the other end. The above model has the potential to ascertain stages of substance use across the dynamic continuum and choose the most appropriate intervention to suit the stage of substance misuse (Table 32.2).

AETIOLOGY: RISK AND PROTECTIVE FACTORS

Substance use does not occur in a vacuum. In vulnerable individuals, substance misuse is produced by the interaction of a drug with genetic, environmental, behavioural, psychosocial and cultural factors (Table 32.3; Boxes 32.1 and 32.2).

The complex mechanisms by which risk and protective factors mediate and modulate development of substance misuse are beyond the scope of this chapter, and interested readers may refer to excellent reviews or textbooks [11,12].

Antecedent and comorbid mental health problems

Community-based longitudinal studies show that depression may predict alcohol dependence and cannabis use [13]. In addition, conduct problems in childhood predict substance abuse and dependence in early adulthood, after controlling for a range of social and other covariates [2]. Similarly, untreated attention-deficit hyperactivity disorder (ADHD) has been shown to be a significant risk factor for development of substance misuse in adolescence and adulthood [14]. The combination of conduct disorder and hyperactivity carries a particularly high risk. The risk of development of substance misuse is high in children exposed to neglect and maltreatment [15,16].

Significant rates of comorbid psychiatric disorders were reported in the community and in clinical samples of young people with substance misuse [17,18], the most common being conduct disorder, major depression, ADHD (with or without comorbid conduct disorder), anxiety disorders [post-traumatic stress disorder (PTSD) and phobias] and bulimia nervosa. Coexisting substance misuse has implications for the onset, clinical course, treatment compliance and prognosis for young people with psychiatric disorders [17,18]. Comorbid substance misuse is the single most important factor that increases the risk of suicide in young people with psychosis or major depression [19].

CONSEQUENCES AND ASSOCIATED FEATURES OF SUBSTANCE MISUSE

A hallmark of substance misuse in adolescents is impairment in psychosocial and academic functioning. Impairment can include family conflict or dysfunction, interpersonal conflict, and academic failure. Associated characteristics such as offending behaviour, other high-risk behaviours and comorbid psychiatric disorders contribute further to risks and impairments. Injecting drug use is rare and only a small minority of young people develop physical dependence. Mortality is high due to accidents, suicides and physical complications of substance misuse. In the UK, volatile substance misuse accounts for 65 deaths per year, which is about 2% of all deaths below the age of 18 years [5].

ASSESSMENT

Information should be obtained from a variety of sources including the young person, parents/other caregivers, general practitioner, school,

Table 32.2 A pragmatic classification of adolescent substance use and the range of interventions. Reproduced from Mirza and Mirza [11].

Stage	Purported motive	Setting	Frequency	Emotional impact	Behaviour	Impact on functioning	Suggested interventions [3]
Experimental stage	Curiosity and risk taking	Alone or with peer group	Occasional at best	Mind-altering effects of drugs are less relevant	No active drug-seeking behaviour	Relatively little, but rarely results in dangerous outcome	Universal prevention (drug education) by Tier 1 services
Social stage	Social acceptance	Usually facilitated by peer group	Occasional but variable, depending on peer group	Mind-altering effects of drugs are clearly recognized and appreciated	No active drug-seeking behaviour	Usually a normative experience. May be associated with significant dangers in rare instances	Universal prevention (drug education) by Tier 1 services
At risk or prodromal stage	Cope with negative emotions or enhance pleasure	Alone or with peer group: mostly on their own	Frequent use	Uses drugs purportedly to alter mood or behaviour	Active drug-seeking behaviour	Impairment in functioning in some areas, but able to hide them by and large	Targeted intervention/ treatment by Tier 2–3 agencies
Stage of harmful use (similar to ICD-10)	Drug use is the primary means of recreation, coping with stress or both	Alone or with an altered peer group	Regular use, despite negative conse-quences	Very important	Active drug-seeking behaviour	Impairment in almost all areas of life and or distress in near and dear	Treatment by Tier 3 agencies
Stage of dependence (similar to ICD-10)	To deal with withdrawal symptoms, and stop craving	Alone	Compulsive use, tolerance and loss of control of use	Very important especially dealing with dysphoria and other withdrawal symptoms	Compulsive drug-seeking behaviour; may engage in acquisitive crimes	Physical and psychological complica-tions; impairment in all spheres of life	Treatment and habilitation by Tier 3 and Tier 4 agencies

ICD-10, *ICD-10 International Classification of Mental and Behavioural Disorders in Children and Adolescents*.

social services, youth justice system or any other social agencies involved. Clinical and research experience shows that young people are generally more reliable informants than might be assumed. The attitude of the clinician should be flexible, empathic and non-judgemental to engage the young person in the assessment process and to obtain a valid estimate of substance use. Explore the young person's leisure activities and gently guide them to talk about the nature and extent of substance use, its context, and its impact on various domains of their psychosocial functioning. This will enable the clinician to determine whether the current pattern of substance use constitutes normative stages of substance use, or meets diagnostic criteria for harmful use or dependence. Detailed exploration of comorbid psychiatric disorders and their relationship to substance misuse would help to formulate a differential diagnosis and treatment plan. Substance misuse is almost

Table 32.3 Risk factors for the development of adolescent substance misuse.

Domain	Risk factor
Neurobiological	Genetic susceptibility to substance misuse Psychophysiological vulnerability (EEG, ERPs) Neurochemical abnormalities (DA, 5-HT, opioids etc.)
Psychological	Depressive disorder Anxiety disorder Early/persistent conduct symptoms, ADHD Physical and sexual abuse Traumatic/stressful life events Early onset of drug use Sensation-seeking traits in personality
Family	Drug use by parents/other family members Family conflict and disruption Inconsistent or harsh discipline Lack of parental expectations about the child's future
Peer group/school	Peer rejection/alienation from peer group Association with drug-using peer group Poor commitment to school Academic failure/underachievement
Social/cultural	Easy availability of drugs Social norms or laws favourable to drug use Extreme economic deprivation Disorganized, anomic neighbourhood

ADHD, attention deficit hyperactivity disorder; DA, dopamine; EEG, electroencephalogram; ERP, Event Related Potential; 5-HT, 5-hydroxtryptamine (serotonin).

Box 32.1 Protective factors

- Close, affectionate parent–child relationship
- Parental monitoring of young person
- Authoritative parenting style
- High educational aspiration/commitment
- Having a non-drug-using peer group
- Good social and interpersonal skills
- Sense of bonding to school or other social institutions (sports club, church, mosque)
- Acceptance of socially approved values and norms of behaviour

always not the only problem and a comprehensive developmental, social and medical history should be undertaken to determine the multiple complex needs across different domains. Particular attention should be paid to the young person's vulnerability, resilience, hopes and aspirations. Evaluating the adolescent's readiness for treatment or stage of change may help determine the initial treatment goals or level of care.

Mental state examination and physical examination

Young people may present with features of intoxication or withdrawal. Recent injecting sites, bloodshot eyes, nicotine stains on fingers, unsteady gait and tremulousness give indications of the extent of substance use. Perceptual abnormalities may suggest a primary psychotic illness or the use of drugs such as cannabis, alcohol, amphetamine or cocaine. Inhaling solvents from the bag may lead

Box 32.2 High-risk groups (based on longitudinal studies)

- Young offenders
- Children of drug-misusing parents
- Children excluded from school/truants
- Young people looked after by local authority
- Young people leaving care
- Young homeless people
- Teenage mothers
- Young people attending mental health services
- Regular attendees of accident-and-emergency services

to a rash around the mouth and nose. Risk of harm to self and others should be systematically assessed, especially in young people with a history of offending behaviour and those with comorbid psychopathology. Psychiatrists should not hesitate to use their hard-won medical skills, and a detailed physical examination including basic neurological examination should always be undertaken. Specific attention should be paid to signs of liver disease, tachycardia and high blood pressure, which may indicate excessive substance use or withdrawal states.

Investigations

Haematological and biochemical investigations like liver function tests are helpful to establish drug- and alcohol-related harm. Testing bodily fluids (urine, saliva, blood) for specific substances should be part of the initial evaluation, especially in inpatient settings and for court-mandated assessments. Most substances – except benzodiazepine, methadone and cannabis – are detectable in urine for a few days only. Considering the above and the potential for adulteration of samples, a negative urine result does not necessarily mean that the young person is not using drugs. A hair test is more reliable as it gives a longer historical profile of drug use (up to 1 month). However, some professionals argue that testing adds little to the verbal reports of substance use in young people, especially when clinicians have managed to nurture a trusting therapeutic relationship with them. There is little evidence at present to recommend repeated testing of bodily fluids to monitor routine clinical treatment.

TREATMENT

The primary goal of treatment is to achieve and maintain abstinence from substance use. While abstinence should remain the explicit, long-term goal of treatment, harm reduction may be an interim, implicit goal, in view of both the chronicity of substance misuse in some young people and the self-limited nature of substance misuse in others. Treatment modalities used are largely psychosocial. Medication is used as an adjunct only, though it may offer a window of opportunity for young people to engage in psychosocial treatment [19,20].

Evidence base for treatment

Reviews of the literature on adolescent treatment outcomes have concluded that treatment is better than no treatment [21]. Naturalistic follow-up of young people in a number of treatment settings in the USA showed decreased substance misuse and criminal involvement, as well as improved psychological adjustment and school performance, one year after treatment [21,22]. Family therapy approaches such as multisystemic therapy [23] and multidimensional family therapy [24] have the best evidence base for efficacy across a number of domains [25], although individual approaches such as cognitive–behavioural therapy (CBT) – both alone and in combination with motivational enhancement – have been shown to be efficacious [26,27]. There is an emerging evidence base for brief motivational interviewing as well [28–30].

Most of the research on psychological treatment comes from the USA, and is not necessarily directly applicable to the UK context, both in terms of the resources required and cultural differences. However, there are significant overlaps between different forms of psychotherapies in both theoretical conceptualizations and therapeutic techniques, and building on existing skills of practitioners working across voluntary and statutory agencies in the UK could prove to be an effective and cost-effective way of delivering evidence-based interventions. Essential elements of a successful treatment programme may include the following:

- An empathic and non-judgemental therapist, who takes painstaking efforts to engage even the 'hard-to-reach' youngster in the treatment process and rekindles the ability to hope and dream.
- A therapeutic process that involves structured and personalized feedback on risk and harm to young people; emphasis on personal responsibility for change; and strategies to increase self-esteem, self-efficacy, practical problem-solving skills and social skills.
- Involvement of family and other 'systems of care' – such as school, judicial system and social services – to address the multiple complex needs of young people.
- A lengthy period of retention in service to ensure good aftercare.

Treatment should be tailored to meet the needs of the individual young person. Integrated mental health and substance misuse treatment should be offered to young people with comorbid psychiatric disorders [29,31,32]. Inpatient treatment is required for a very small minority: those with severe and chaotic substance misuse; repeated failed community detoxification; intravenous drug use with complications; and severe mental illness and risk of self-harm. Variables consistently related to successful outcome are treatment completion, low pre-treatment substance use, and peer and parent social support [21]. Other factors predictive of outcome are involvement of family, use of practical problem-solving, and provision of comprehensive services such as housing, academic assistance and recreation [26].

Role of child and adolescent mental health services (CAMHS)

Despite the significant expansion of specialist substance misuse services over the past decade, many youngsters still do not receive adequate treatment, and there are ongoing debates regarding the role of CAMHS in adolescent substance misuse Professionals working in CAMHS have an unrivalled opportunity to play a significant role in the early identification and treatment of substance misuse, including children of substance-misusing parents and other high-risk groups. Specific treatment of 'core' mental health problems such as depression, eating disorders, ADHD and PTSD is a primary role of the specialist CAMHS [33]. CAMHS professionals could help develop multi-agency treatment services and train other professionals in evidence-based interventions.

CONCLUSIONS

The notion of a drug-free society is almost certainly a chimera. Young people have always used substances to change the way they see the world and how they feel, and there is every reason to think they always will. However, early identification and comprehensive treatment could help to reduce distress and prevent further deterioration. Everything that is done to help troubled and troublesome children should be informed by a sense of history, a reflective awareness of current value systems, economic and social factors, and by a mature and balanced judgement of what is possible and what is not. Integrative, multi-agency treatments addressing a range of ecologically valid aetiological factors have the potential to engender a culture of therapeutic optimism.

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Early-Onset Bipolar Disorder

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INTRODUCTION

Early-onset bipolar disorder (EOBPD) (i.e. onset before 18 years) is a serious psychiatric disorder associated with social and academic difficulties and suicidality. Recently there has been increased recognition of this disorder but no clear consensus on definition.

DIAGNOSTIC CRITERIA

The *ICD-10 International Classification of Mental and Behavioural Disorders in Children and Adolescents* (ICD-10) criteria [1] for bipolar disorder require at least two episodes of significantly disturbed mood and activity with, on occasion, either mood elevation (mania or hypomania) and increased activity or depression with decreased activity and energy. The *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition – Text Revision* (DSM-IV-TR) criteria [2] for bipolar disorder differ slightly, requiring a manic episode – an elevated, expansive or irritable mood lasting 1 week or shorter if hospitalization is required. By convention there are two subtypes of bipolar disorder – bipolar I, where the patient has had at least one manic or mixed episode; and bipolar II, where the patient has had one or more episodes of both major depression and hypomania, but no manic or mixed episodes.

CLINICAL CHARACTERISTICS

There is debate, particularly in the USA, about the clinical presentation of EOBPD, with some authorities recognizing EOBPD as presenting with:

1. chronicity with long episodes;
2. predominantly mixed episodes and/or rapid cycling;
3. prominent irritability; and
4. high rate of comorbid attention deficit hyperactivity disorder (ADHD) and anxiety disorders.

There two main diagnostic issues, which are at variance with the diagnostic practice for adult bipolar disorder (BPD) – firstly, whether elevated/expansive mood is required for a diagnosis of mania, or whether irritability alone is sufficient; and secondly, whether EOBPD is characterized by a chronic, non-episodic course, rather than an illness with manic episodes as required in DSM-IV-TR.

Early-onset bipolar disorder can be conceptualized as narrow, intermediate or broad phenotypes [3]. Those with the narrow phenotype, or BPD type I or II as described in DSM-IV-TR [2], have recurrent periods of major depression and mania or hypomania. The category BPD not otherwise specified (BPD NOS) is reserved for children who fail to meet the duration criteria of 4–7 days required to fulfil the DSM-IV-TR criteria for hypomania or mania. Children presenting with severe irritability, affective storms, mood lability, severe temper outbursts, symptoms of depression, anxiety or ADHD-like symptoms – poor concentration and impulsivity – with or without clear episodicity, are regarded as having the broad phenotype. Recent findings point to a continuum of symptoms and severity from BPD NOS, BPD II to BPD

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I with elevated mood being a common feature throughout [4].

EPIDEMIOLOGY

The peak onset of bipolar disorder is in the 15–19-year-old group, with males and females equally represented. Retrospective studies in adults with BPD report that over 50% of patients experience the onset of their BPD before 20 years of age, and between 10 and 20% report the onset before 10 years of age [5]. The initial presentation may be one of depression; around 20–30% of depressed children, particularly those with psychosis, a family history of BPD and/or pharmacologically induced mania, eventually develop BPD [6].

A community school survey of older adolescents (14–18 years) showed the lifetime prevalence rate to be around 1% [7], although only 0.1% had mania. Most of the identified cases had hypothymia or cyclothymia. Manic symptoms are common in up to 13% of the school population of 14–16-year-olds; however, this figure is reduced to 0.6% if severity and impairment criteria apply [8]. In the Great Smoky Mountains Study of children aged 9, 11 and 13, no cases of BPD type I were found [9]. Subsequent studies focused on those children with chronic irritability and hyperarousal, designated as having 'broad BPD phenotype' or severe mood dysregulation (SMD). The lifetime prevalence of SMD in children and adolescents aged 9–19 was 3.3%, or 1.8% with severe impairment [10]. However, although SMD appears to be part of the affective spectrum with SMD at age 10, predicting depressive disorder in early adulthood, it did not predict BPD.

ASSESSMENT

Semistructured interviews, such as the Kiddie Schedule for Affective Disorders and Schizophrenia – Present and Lifetime (K-SADS-PL) [11], and for children the Washington University in St Louis-Kiddie Schedule for Affective Disorders and Schizophrenia (WASH-U-KSADS) [12] reliably elicit operationally defined symptoms and can be recommended for use in both research and clinical settings. The Young Mania Rating Scale (YMRS) [13] is commonly used in research to assess the severity of manic symptoms, and assess treatment response. However, the YMRS is not a diagnostic instrument.

DIFFERENTIAL DIAGNOSIS

The symptom overlap between ADHD and EOBPD can create diagnostic problems, particularly in the young, where there are high rates of comorbidity – 60–90% according to some [14]. However, the symptoms of grandiosity, elated mood, flight of ideas and decreased need for sleep reliably differentiate the two. Bipolar disorder can be associated with a sudden onset of severe behavioural disturbance. Such disturbance contrasts with the usually longer-standing conduct disorder. A family history of affective disorder rather than conduct or personality disorder may aid diagnosis. In children, mood instability, and irritability associated with pervasive developmental disorders need to be noted [15], while in adolescence affective instability seen in cases of borderline personality disorder can cause diagnostic confusion [16]. In the latter case there may well be considerable overlap, with reports of 15% of patients with bipolar disorder having borderline personality disorder [17]. Psychosis in adolescence, particularly if florid with mood-incongruent hallucinations and thought disorder, has been misdiagnosed as schizophrenia [18]. Factors in favour of a diagnosis of schizophrenia include: premorbid personality abnormalities, schizotypal personality disorder, a family history of schizophrenia, and an insidious onset of psychosis. Mania needs to be distinguished from drug-induced states secondary to drug misuse or, rarely, from medical treatments such as steroids.

LONGITUDINAL COURSE AND PROGNOSIS

An important step in judging the validity of EOBPD is the stability of the diagnosis over the short and long term. The EOBPD phenotype has been reported to be reliable, with stability over follow-up assessments at 6 months and 1, 2 and 4 years. High rates of chronicity and relapse were found during a 4-year follow-up period despite community treatment [19].

Although symptoms of EOBPD appear stable over time [20], EOBPD has not yet been

shown to progress into the classic adult BPD. The evidence is not sufficient to indicate that EOBPD is continuous with adult BPD. The COBY study (Course and Outcome of Bipolar Youth) [21] of 413 youths (aged 7–17 years) with bipolar I disorder ($n = 244$), bipolar II disorder ($n = 28$) and bipolar disorder not otherwise specified ($n = 141$) found that at 2.5 years after the index episode, 81.5% of the participants had fully recovered. However, 1.5 years later 62.5% had a syndromal recurrence, particularly depression. Manic symptomatology, especially syndromal, was less frequent. Twenty-five percent of youths with bipolar II converted to bipolar I, and 38% of those with bipolar disorder not otherwise specified converted to bipolar I or II. Overall the outlook is concerning and emphasizes the seriousness of this diagnosis in this age group.

SUICIDE

Bipolar disorder is a risk factor for suicide. Adolescents with bipolar disorder have higher rates of completed suicide [22] and attempted suicide [23]. Suicide attempts are associated with older age, depressive episodes, mixed states and psychotic features [24]. Comorbid substance abuse, panic disorder and past histories of suicide attempts and physical or sexual abuse add to the risk profile.

TREATMENT

The treatment of early-onset bipolar disorder requires a multimodal approach. An assessment of comorbid disorders such as substance abuse and conduct disorder needs to be undertaken, including an appraisal as to whether these are mood dependent. Comorbid disorders may need treatment in their own right. The treatment plan clearly needs to take account of the developmental level of the child and adolescent and the differing age presentations of bipolar disorder (Table 33.1).

The treatment of bipolar disorder can be divided into two stages: acute treatment of mania or depression, and prophylaxis.

Acute phase

There is an increasing trend to use atypical antipsychotics in children and adolescents, both in the acute manic phase and in the longer-term as mood stabilizers. With the recent US Food and Drug Administration (FDA) indication of risperidone, aripiprazole, quetiapine and olanzapine for the treatment of bipolar youth, the atypical antipsychotics are rapidly becoming a first-line treatment option. However, with the exception of aripiprazole these agents are associated with adverse effects such as increased appetite, weight gain, lipid abnormalities and a risk of type II diabetes mellitus.

Expert guidelines on the treatment of paediatric bipolar disorder [25] recommend the use of mood stabilizers and or atypical antipsychotics (Figures 33.1 and 33.2). A combination of mood stabilizers and atypical antipsychotics is often advocated if there is no response to single-medication treatments. However, some argue against this, as trials of sodium valproate [26] and oxycarbazepine [27] have been negative. The choice of medication depends on the phase of the illness, presence of psychosis, presence of rapid cycling, risk of side effects, and, crucially, patient and family acceptance. Atypical antipsychotics are recommended for treating psychotic symptoms but they also act as mood stabilizers. Premature

Table 33.1 Evidence for medication treatment for child and adolescent bipolar disorder.

Medication	Evidence level
Lithium	A&B
Valproate	B&C
Carbamazepine	B
Oxcarbazepine	–
Topiramate	–
Clozapine	C
Risperidone	A (FDA approval)
Olanzapine	A (FDA approval)
Quetiapine	A (FDA approval)
Aripiprazole	A (FDA approval)
Lamotrigine	C
Ziprasidone	B&C

Level A data: child randomized controlled clinical trials.
Level B data: adult randomized clinical trials.
Level C data: open trials and retrospective analyses.
Level D data: case reports and expert opinion to recommended current clinical practices.

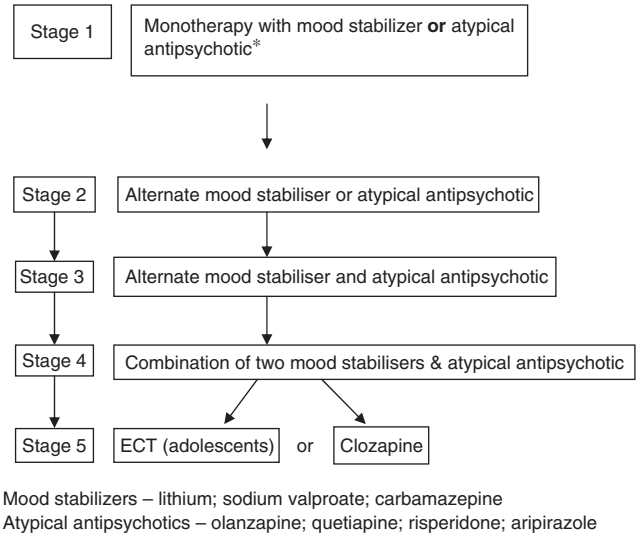

Figure 33.1 Bipolar disorder type I (BPD-I), manic, mixed, without psychosis. ECT, electroconvulsive therapy.

discontinuation of antipsychotic medication leads to a recurrence of psychotic symptoms in a large percentage of cases [28].

Opinion is that medication tapering or discontinuation be considered if the patient has achieved remission for a minimum of 12–24 consecutive months. However, for many patients long-term or even life-long pharmacotherapy might be indicated.

Treatment of depression in bipolar disorder

The first-line treatment for milder depression should be psychological (e.g. cognitive–behavioural therapy). In patients with bipolar disorder, selective serotonin reuptake inhibitors (SSRIs) are recommended antidepressants, but these may need to be used alongside a mood stabilizer. The use of lamotrigine has been shown to be effective in children and adolescents [29]. For severe depression with psychotic symptoms an antipsychotic, such as risperidone, with an antidepressant and a mood stabilizer would be appropriate. Electroconvulsive therapy (ECT) is recommended in severe psychotic depression, especially if there is a risk of suicide.

Psychological treatments

For bipolar disorder, adjunctive psychotherapy enhances the symptomatic and functional outcomes over a 2-year period, although there is less evidence for early-onset cases [30]. Treatments that emphasize medication adherence and early recognition of mood symptoms such as psycho-education have stronger effects on mania, whereas treatments that emphasize cognitive and interpersonal coping strategies, such as CBT and family therapy, have stronger effects on depression.

Family-focused therapy for adolescents with EOBPD (13–17 years) (FFT-A) involving 21 sessions over 9 months and follow-up at 2 years, showed that FFT-A was associated with a faster recovery from depression compared to brief psycho-education [31]. High expressed emotion (EE) attitudes among parents are generally associated with an increased likelihood of relapse in EOBPD. This highlights the importance of a family approach with FFT-A, which results in a greater reduction in depressive and manic symptoms in high EE families [32]. There is a preliminary report of an open 1-year trial of dialectic behavioural therapy with family and

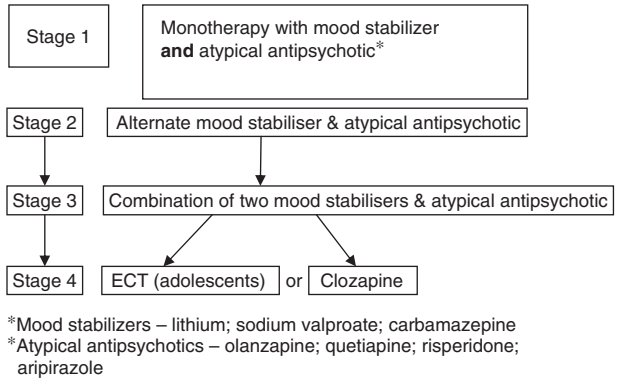

Figure 33.2 Bipolar disorder type I (BPD-I), manic, mixed, with psychosis. ECT, electroconvulsive therapy.

individual components for bipolar disorder [33], which may be a promising treatment.

Overall, there are clear limitations to present treatments, with one study finding that participation in community treatment (including mood stabilizers) did not improve outcome at 2 years [20].

Refractory cases

Clozapine is recommended for refractory bipolar disorder but with caution due to the side effects. Require regular monitoring with blood tests is essential.

ECT is rarely used in adolescents, but case series and reports support its use in severe life-threatening psychotic depression or treatment-resistant mania. The response rate for psychotic disorders is 50–60% [34]. ECT requires appropriate consent, and given the mental state of the patient this is very likely to be under appropriate legislative powers.

SCHIZOAFFECTIVE DISORDER (SA)

The concept of schizoaffective disorder remains problematic. Indeed, there is little evidence for temporal stability for the diagnosis of SA in this age group [35]; however, genetic studies [36] show that broadly defined schizoaffective disorder, bipolar type, is genetically homogeneous. Nevertheless, there are questions over the distinction between schizophrenia and bipolar disorder with psychosis on the grounds of a largely shared genetic basis, with the exception of copy number variations [37].

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34

Emerging Personality Disorder

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DEFINITIONS

Temperament

Temperament includes individual differences in self-regulation, affect, attention and activity. These traits have a biological, partly genetic basis whilst at the same time their development is affected by maturation and life experience [1]. Temperamental features are noted more often in younger children such as babies or toddlers, who may be described as having a 'sunny', 'placid' or 'restless' temperament.

Personality

Personality is a more complex, multidimensional construct than temperament, and this may be partly why it is more likely to be applied to older children and adolescents. Obvious personality traits such as neuroticism and extraversion can be readily noted as well as other processes associated with different personality types, such as coping styles, attachment styles, motives and goals [2].

Current thinking favours consideration of temperamental style and personality traits together 'Because of the significant commonalities between temperament and personality traits' (Ref. [3], p. 182).

PERSONALITY DEVELOPMENT

While the exact definitions of both temperament and personality have evolved over time [3], it is currently recognized that both will develop under the influence of interactions and transactions between constitutional, genetic and environmental factors [1].

Research demonstrates both continuity and change in personality trait development. Thus, while childhood personality traits are moderately stable by the age of 4 years, developmental changes continue into adult life – though only moderate change is to be expected after the age of 50 years [4].

CHILDHOOD PERSONALITY TRAITS AND ADULT OUTCOMES

Most research has focused on pathological outcomes with the evidence linking certain childhood personality traits and behaviours with specific adult outcomes. For instance, children showing early neurocognitive problems (including early difficult temperament) and a 'life course persistent' trajectory of antisocial behaviour may develop conduct disorder and antisocial personality disorder in adult life [5,6]. A small number of high-risk children on this trajectory start offending earlier, commit more violent offences and have higher levels of recidivism [7–9]. This group has a substantial genetic risk of psychopathy and shows 'callous unemotional' personality traits found in adult psychopaths [10,11].

Recent neuroscientific evidence has increased understanding of some childhood personality traits. Brain studies suggest structural differences in the brains of children with callous unemotional traits [12]. There are also suggested links between psychophysiological features, such as skin conductance measures, in early childhood and later

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psychopathy, as measured by self-report, at 28 years of age [13].

However, longitudinal research looking at outcomes for normal developmental personality traits is still lacking although the 'Big Five' model (Table 34.1) may provide a framework against which positive and negative trait outcomes could be measured [14].

PERSONALITY ASSESSMENT AND PERSONALITY DISORDER IN CLINICAL PRACTICE

Why assess childhood personality?

Clinicians do not routinely assess infant temperament or childhood personality. There are concerns about applying pejorative diagnostic labels to young children, alongside a concern that focusing on child temperament may exert pressure on clinicians to make diagnoses of personality 'disorder' in later adolescence. There are also groundless fears that personality traits are unchangeable, whereas they evolve and develop through transactions between individuals and their environments [3].

Even where underlying personality/temperamental traits do contribute to presenting problems, interventions are likely to focus on their behavioural expression. For instance, interventions for childhood oppositional defiant behaviour may take the form of parenting programmes rather than beginning with an assessment of the child's inherent temperament.

This lack of personality assessment is regrettable as it has been suggested that treatment effectiveness may well be maximized where interventions are tailored to the personality of young participants [3].

Definitions of disorder: diagnostic issues

Use of diagnostic criteria from either the *Diagnostic and Statistical Manual of Mental Disorders, 4th edition, Text Revision* (DSM-IV-TR) or the *International Classification of Diseases, 10th revision* (ICD-10) allows the clinician to codify any presenting signs of personality dysfunction in children and adolescents Within DSM-IV-TR it is possible for a child or younger adolescent to be given a diagnosis of a personality disorder, although Antisocial Personality Disorder (ASPD) can only be diagnosed at age 18 years old [15].

In community-based clinical practice (Child and Adolescent Mental Health Services, or CAMHS), it may be unusual for a child or young person to present with the full criteria for any one personality disorder. However, the presence of subthreshold (a few) traits of a personality disorder does not mean that there is no cause for concern. Rather this situation should alert the clinician to the need for follow-up and to reassess the child or young person to provide or to modify treatment interventions.

Table 34.1 'Big 5' and possible Positive and Negative Outcomes [14].

Trait	Positive outcome	Negative outcome
Extraversion	Social competence Promotes good health; better romance/ long-term relationships	Antisocial behaviour; callousness
Neuroticism	Conscience development; guilt when expected	Poor relationships; relation conflict; relation abuse; less competent parenting; risk for unemployment
Conscientiousness	School adjustment; educational and occupational achievement; job performance	Obsessive
Agreeableness	Social competence; positive parenting; responsible parenting	Exposure to risks
Openness	Exploring friendliness; academic achievement	Exposure to risks

In contrast, more complex cases seen in specialist services (Tier 4 NHS) may fully satisfy criteria for various personality disorders. Co-morbidity for a range of other psychiatric and behavioural disorders is also the norm in samples of children (and also adults) referred to specialist services such as adolescent psychiatric units and forensic services.

In cases where there is concern about a child's personality traits or an emerging personality disorder, an assessment should be done, covering all aspects of the child's development including temperament, personality and family life (**17;** Figure 34.1). A family assessment of children at risk of ASPD or psychopathy is particularly important given the role of family risk factors, including parental mental illness and criminality, in their development [5,6,9,14].

Lessons from clinical practice

The following childhood precursors of personality disorders can be considered in assessment:

Paranoid Personality Disorder: This is uncommon in adolescence. Those presenting with suspicious, distrustful or apparently paranoid features should be assessed to exclude drug abuse, particularly cocaine dependency, and communication problems such as a hearing impairment. Delinquent adolescents who have criminal records may also present as suspicious, distrustful and anti-authoritarian on the basis of negative experiences with care and courts systems.

Schizoid Personality Disorder: Some of the diagnostic criteria for this disorder (such as choosing solitary activities, few close friends, emotional coldness, etc.) overlap with and should be distinguished from other childhood conditions such as shyness, intellectual disability and autistic spectrum disorders.

Schizotypal Personality Disorder: Children with schizotypal features usually present to services with queries about autistic spectrum disorders or incipient schizophrenia. These should be excluded with a full mental state assessment and developmental history. Some schizotypal features such as bizarre behaviour and facial expressions can be seen in children with serious learning disabilities who are on the autistic spectrum.

Schizotypal children may show quasi-psychotic features such as ideas of reference, which just stop short of delusions. They may also feel that they have magical powers over others; in children from certain ethnic minority families, these individual, distorted magical beliefs need to be distinguished from widespread cultural beliefs such as voodoo, speaking in tongues, shamanism, etc. This may be important in the management of cases with suspicions of child abuse or where children are seen as witches, being possessed by the devil and needing exorcism, etc.

Anti-Social Personality Disorder (ASPD): In practice, children with persistent, severe conduct disorder will have a wide range of other social, emotional, educational and intellectual difficulties all of which need a full multidisciplinary assessment [16,17]. **Co-morbidity** for psychiatric disorders is the norm for children with persistent, severe conduct disorder including pervasive developmental disorders and other disabilities [18]. Children of parents with an ASPD or psychopathy traits may be at greater genetic risk of developing a personality disorder.

An assessment of the risk of that child developing an ASPD or psychopathy should be undertaken. These cases usually involve child protection concerns and complex developmental disorders so a holistic assessment covering all aspects of the child's needs, family functioning and parenting capacity should be undertaken (see Figure 34.1). Parental ASPD or psychopathy may also have major implications for the parenting, placement and care of high-risk children [19].

Borderline Personality Disorder (BPD): Adolescents with BPD may be comorbid for other personality disorders and psychiatric disorders – a common feature amongst disturbed forensic populations [8]. The life course outcome for individuals with BPD suggests that the dysfunctional behaviour will wane in their 30s and 40s. Meanwhile, clinical experience shows that these individuals can create havoc amongst their own families, within agencies and in society. Hence, identification of adolescents at risk of acquiring BPD is important in preventing offending, incarceration and subsequent poor parenting [19].

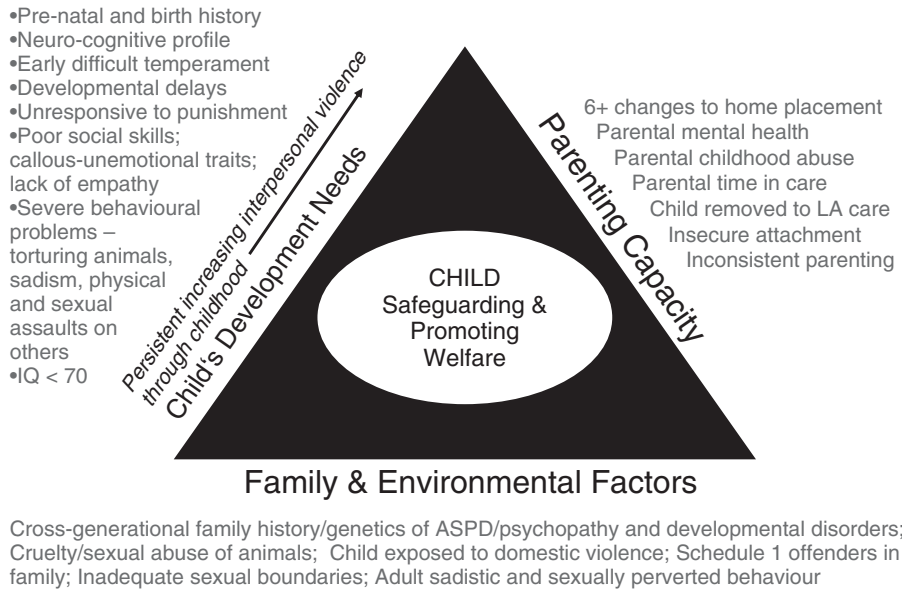
•Pre-natal and birth history
•Neuro-cognitive profile
•Early difficult temperament
•Developmental delays
•Unresponsive to punishment
•Poor social skills; callous-unemotional traits; lack of empathy
•Severe behavioural problems – torturing animals, sadism, physical and sexual assaults on others
•IQ < 70
Persistent increasing interpersonal violence through childhood
Child's Development Needs
CHILD Safeguarding & Promoting Welfare
Parenting Capacity
6+ changes to home placement
Parental mental health
Parental childhood abuse
Parental time in care
Child removed to LA care
Insecure attachment
Inconsistent parenting
Family & Environmental Factors
Cross-generational family history/genetics of ASPD/psychopathy and developmental disorders; Cruelty/sexual abuse of animals; Child exposed to domestic violence; Schedule 1 offenders in family; Inadequate sexual boundaries; Adult sadistic and sexually perverted behaviour

Figure 34.1 NCATS Emerging Severe Personality Disorder (ESPD) assessment triangle [17].

Histrionic Personality Disorder: Histrionic Personality Disorder does not present frequently to CAMH services until later in adolescence. Nevertheless, the shallow and transient nature of emotional relationships achieved by individuals with Histrionic PD suggests that identification and treatment of adolescents at risk of this disorder would be beneficial for them and their families of procreation.

Narcissistic Personality Disorder: Few adolescents present to clinical services solely with marked narcissistic traits. However, narcissistic attitudes may be seen in other PDs, such as the adolescent sex offender with an ASPD who also has a strong narcissistic sense of entitlement (to abuse whom he pleases) but who fails to fulfil criteria for Narcissistic PD.

If narcissistic traits persist after adolescence into later life, such individuals may have difficulties in adjusting to the inevitable limitations of the ageing process. In some adult individuals this may present as a 'Peter Pan' or denying attitude towards growing older with attempts to recapture a mythical youth through surgery, inappropriately youthful dressing, etc.

Avoidant Personality Disorder: There are overlaps between Avoidant Personality Disorder and many other disorders such as social phobia, Dependent Personality Disorder, and paranoid, schizoid and schizotypal disorders. Caution is needed in applying this diagnosis to children and young people, some of whom may have acculturation problems following immigration or may simply be shy adolescents passing through a normal developmental phase.

Dependent Personality Disorder: Crucially, the degree of dependency on others should be age inappropriate and situation inappropriate. For instance, adolescents may expect their parents to take all decisions about which friends they should have and how they should spend their free time. With younger children who may show apparent traits of Dependent Personality Disorder, great caution should be taken in applying the diagnosis because dependent behaviour may be developmentally appropriate in many younger children.

Some victimized children may show signs of an attachment disorder, aspects of which may overlap with Dependent Personality Disorder. Alternatively, the child's cultural norms may be such that

acquiescent or passive behaviour could be confused with dependency.

Obsessive-Compulsive Personality Disorder: Obsessive-Compulsive Personality Disorder (OCPD) should be distinguished from Obsessive-Compulsive Disorder (OCD). The presence of true obsessions and compulsions differentiates OCD from OCPD, where gaining control over situations is a key element.

In practice, many young children may pass through an age appropriate phase where they show apparently obsessional characteristics such as arranging their toys or possessions in a particular order, lining up their food on the plate to eat in a set sequence, etc. These behaviours are usually age appropriate and will pass in time. Adolescents with hobbies that, for instance, involve collecting and categorizing items and acquiring extensive knowledge of a particular hobby topic, such as football, may present as obsessive or 'nerdy' but will usually grow out of this phase and move on to another interest, likely to be less obsessively followed.

Personality Disorder Not Otherwise Specified (NOS): This diagnosis is useful for those who do not completely fulfil criteria for a particular PD but show one or more traits of several PDs.

CONCLUSIONS

Natural pathways exist that take the infant through a series of physical, emotional, cognitive and social developmental stages towards childhood, adolescence and adult life [20]. Links appear to exist between infant temperament, childhood personality traits and adolescent personality styles.

Empirical research has not yet tracked these links across childhood and adolescence with detailed reference to a wide range of personality traits and outcomes including diagnoses of adult personality disorders. An exception to this is that adult outcomes for children with certain personality traits and behavioural profiles, such as conduct disorder, are now reasonably well mapped [5].

Personality disorders carry a burden of psychopathology, relationship and parenting problems in adult life [19]. Hence, it seems advisable for mental health assessments of children and adolescents routinely to include reference to normal personality traits and to any signs of emerging personality disorders.

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Section 6

Assessment

35

Diagnostic Classification: Current Dilemmas and Possible Solutions

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WHAT'S NEW

- A draft for the DSM-5 classification scheme has been prepared and posted on the internet.
- It provides more developmental considerations, such as psychosis risk and adult ADHD.
- There are some new categories of particular relevance to child psychiatry, including temper dysregulation, non-suicidal self-injury and a callous-unemotional type of conduct disorder.

Developments in classification can sometimes have a major impact on the clinical world. The *Diagnostic and Statistical Manual of Mental Disorders, Third Edition* (DSM-III) and *International Classification of Mental and Behavioural Disorders in Children and Adolescents, Ninth Revision* (ICD-9) were both great steps forward for the ability of clinicians to communicate effectively with each other and the world of research. Inter-rater reliability started to be good enough for effective audit, sharing of clinical lessons and establishing research series [1]. The revised third edition of DSM (DSM-III-R) and DSM-IV refined the process, added new categories and responded to research findings [2]. ICD-10 came into greater harmony with DSM and developed algorithmic criteria for research purposes. They were not, however, conceptual leaps forward. Revisions into DSM-5 and ICD-11 are now underway, so it is timely to consider what they have to achieve.

The next big conceptual advance in classification is likely to be the establishment of pathophysiologically grounded diagnoses. We aspire to use advances in neuroscience to establish psychiatric illnesses that can be assessed objectively and treated rationally. Some might argue that the time for this has come. Should we use our knowledge – for instance of dopamine changes in schizophrenia, or frontal and striatal underactivation in attention deficit hyperactivity disorder (ADHD) – to redefine conditions in a way that would allow diagnosis by neuroimaging? The answer must be 'not yet'. Our neurobiological knowledge is based on group studies; within groups there is considerable heterogeneity and between diagnostic groups there is considerable overlap. The implications for individual diagnosis will need better understanding before a radical change is feasible.

The groups working on DSM-5 include some whose purpose is to consider whether a preliminary regrouping of disorders is feasible on the basis of present neuroscience knowledge. Should we, for instance, group ADHD with the disorders of addiction on the basis of neurochemistry, rather than with disruptive behaviour disorders on the basis of longitudinal course, or with neurodevelopmental disorders on the basis

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of neuropsychological findings? Even in this well-studied condition, controversy is strong. It seems unlikely that a broad consensus can be achieved in the near future.

If a new basis for classification has not yet arrived, is there still a need for revising the classification schemes? Any revision brings penalties. Knowledge and practice based on existing entities can be outdated unnecessarily, and apparently trivial changes in wording can have marked effects on the prevalence of conditions. I would argue that the current schemes, valuable though they have been, contain some flaws that limit their usefulness and can be amended.

- We have increasingly learnt that many syndromes behave as continua in the population. As a result, cut-offs are often arbitrary, reliability is low around the point of cut-off, and there is scope for a runaway expansion of diagnoses.
- The relationship between symptoms and impairment of function is often remote [3]. In the absence of clear points that delimit the pathological from the normal, we often have to require impairment as a necessary condition for diagnosis. There are disadvantages for prevention, and it is rather offensive if a person who has compensated well should no longer be considered as having the condition.
- For many conditions the threshold for diagnosis is set by a fixed number of symptoms. This has proved useful, but takes too little account of possible differences between the genders or between different levels of maturity [4].
- Many childhood conditions have proved to persist into adult life. The changing circumstances and demands of adulthood mean that different aspects of the condition should be given more weight in the diagnostic criteria. Diagnostic criteria may therefore have to change in their nature and number in order to do justice to developmental changes.
- The features of an adult-type disorder in childhood need recognizing – but they may be very similar to those of other conditions. For instance, the antecedents of schizophrenia may include clumsiness, neuropsychological changes, social incompetence and odd thinking. These features, however, may also be seen in other neurodevelopmental problems, including autism spectrum. This could result in a mistaken diagnosis of 'pre-schizophrenia' or 'psychosis risk' for a child with a different condition [5].
- Most children with one DSM diagnosis also have others. This mirrors reality: problems in young people's mental life are often multiple. The resulting proliferation of diagnoses in the individual case is conceptually untidy, does not allow for some problems being complications of an underlying disorder, and makes for difficulties in patients' understanding and in the commissioning and planning of services. 'Comorbidity' between disorders is a topic of active research but we do not yet have the clarity of understanding that would let us cope rationally with these overlaps [6].
- The sheer number of categories available, the overlap between some of them, and the possibility of impaired people meeting no diagnosis, all limit practicality. They deter non-specialists from using the psychiatric scheme. ICD-10 has avoided some of this difficulty by a system of descriptive and exclusion categories that create an expectation of a single diagnosis for most people. On the other hand, single diagnoses can lead to an under-recognition of non-classical features of disorder. Obsessive-compulsive problems, for instance, may need recognition even in a person in the autism spectrum.

The approaches to solving some of these difficulties can be illustrated from specific disorders: the so-called paediatric bipolar disorder, the presence of ADHD in adult life, the presence of post-traumatic stress disorder in early life, the 'lumping' or 'splitting' of conditions within the autism spectrum and the description of syndromes involving self-harm (Table 35.1).

PAEDIATRIC BIPOLAR DISORDER

The traditional definition of bipolar disorder includes a requirement for distinct periods of altered mood with the key qualities of mania – euphoria, grandiosity and irritability. In adult psychiatry, however, it has become plain that many severely affected people have a condition involving very rapid and frequent cycles of mood.

Irritability is a very common problem of child mental health. It often challenges both parents and schools and is a very frequent reason for

Table 35.1 Some issues in current classification.

Problem	Example	DSM-5 proposal
Unvalidated distinctions	Autism-related conditions	Severity dimensions within one autism spectrum
Gender/age adjustment	Adult ADHD Child PTSD	New age-specific criteria
Frequent comorbidity	Irritability	Cross-cutting dimensions
Many categories	Anxiety-based disorders	Superordinate clustering
Heterogeneity within categories	Self-harm	New category of non-suicidal self-injury
NOS categories	Paediatric bipolar disorder	New condition of temper dysregulation

ADHD, attention deficit hyperactivity disorder; DSM-5, *Diagnostic and Statistical Manual of Mental Disorders, 5th edition* (draft); PTSD, post-traumatic stress disorder.

referral. The symptom of irritability, however, is a feature of several different disorders. In order to do justice to the problems of children with intense and volatile moods, the proposal has been made that the diagnosis of bipolar disorder should be expanded in children, to include non-episodic states of angry outbursts, not necessarily accompanied by euphoria [7]. The result, however, has been a disquietingly large increase in the rates of diagnosis and in the prescription of neuroleptics and mood stabilizers. The issue has become highly controversial. DSM-IV allowed this expansion of diagnosis, partly because the wording of items was not adapted to children, and partly because the subcategory of 'not otherwise specified' allows bipolar disorder to be diagnosed even in the absence of defining criteria.

The draft of DSM-5 therefore proposes a new disorder: 'temper dysregulation disorder with dysphoria'. This is intended to provide a conceptual home for severely affected children who combine a persistent mood of misery or anger with very marked irritable outbursts. There is some empirical support for such a category, for instance in the tendency of the condition to persist in the same form over time, and neuroimaging distinctions between children with 'severe mood dysregulation' and those with classic bipolar disorder. Nevertheless, such a category may prove to have disadvantages. It will usually coexist with other conditions (e.g. oppositional disorder, depression or dysthymia). It does not yet meet all the stringent requirements for a new disorder. The name of 'temper dysregulation' could invite a pathologizing of normal childish tempers – even though the criteria of the new disorder are written to describe only a high level of severity. Field trials will therefore be very useful to assess the robustness of the new diagnosis in practice and its knock-on effect on other conditions.

There is a general issue of how to deal with common symptoms that cut across existing diagnostic categories. My own view is that this is best dealt with by the use of cross-cutting dimensions that allow the clinician to describe not only the presence but also the severity of clinical problems such as irritability, anxiety and social impairment. It remains to be seen how far, and in what way the revised DSM will cope with this. Such a revision could be a significant advance in the concepts available to clinicians.

ATTENTION DEFICIT HYPERACTIVITY DISORDER (ADHD) IN ADULTS

There have been advances in knowledge, both from surveys in adult populations and from follow-up studies into adult life of people with ADHD. They have indicated that people can be impaired by symptoms of ADHD that fall short of the full criteria for the diagnosis. The DSM-5 work groups have responded to this with a review of the studies, and a consequent relaxation of the number of criteria that have to be met by people over the age of 18. There are more examples provided of inattentive, overactive and restless behaviours that can be shown by adults. The requirement for an onset of disorder before the age of 7 has also been relaxed, and now expects only the presence of symptoms (not necessarily impairment) before the age of 12 years. The practical effects are likely to be a substantial increase in the recognition and treatment of ADHD in adult life.

Other ADHD issues are unresolved. The question of different criteria for males and females has not been satisfactorily answered. The different subtypes (inattentive, hyperactive-impulsive and combined) have failed to generate significant differences between them in course and associations; so there would be a case for abolishing the distinctions. On the other hand, 'inattentive subtype' is popular with clinicians. The apparent lack of validity may stem from the inclusion of cases who are only just subthreshold for hyperactivity-impulsivity. A more strictly defined inattentive subtype could be useful. Such children may be sluggish in their cognitive tempo, and may have a course characterized more by educational and occupational failure than by the conduct problems associated with hyperactivity. Systematic review, however, failed to find a clear basis for a cut-off, and the issue remains unresolved – as it was in DSM-III, DSM-III-R and DSM-IV.

DISRUPTIVE DISORDERS

The current distinction between oppositional and conduct disorders seems likely to remain, because of the affective components in the definition of oppositional disorder (e.g. anger and spitefulness). It remains to be seen whether this will still be necessary if the affective component proves to be satisfactorily described by 'temper dysregulation disorder with dysphoria' (or whatever name is eventually agreed for conditions of severe emotional dysregulation).

The callous and unemotional aspects of some children and adults with conduct disorder have been increasingly recognized in recent research. A subcategory or specifier within conduct disorder will therefore be included in the DSM-5, to provide a standard way of diagnosis. The name, however, is controversial. On the one hand 'callous-unemotional' sounds as if it could be stigmatizing language and hard to explain to families. On the other hand, the 'C-U' phrase is widely used and easy to understand.

SELF-INJURY

Harming oneself is not a disorder, but a feature of other conditions – including depression and borderline personality disorder (BPD). There is, therefore, a problem for the diagnostician when the behaviour of self-harm is not accompanied by a diagnosable condition. This can lead to the loose use of a diagnosis such as BPD; and it can sometimes lead to inappropriate practice. Many forms of self-injury do not include suicidal ideation but may lead to inappropriate hospital admission. A new category is therefore being proposed – non-suicidal self-injury – to provide a way of describing the infliction of superficial skin injury without the intent to endanger life. It is a way of coping with the heterogeneity of self-harming actions.

FURTHER PROGRESS

The development of the fifth edition of the *Diagnostic and Statistical Manual* of the American Psychiatric Association has reached the stage of draft proposals. This chapter cannot, of course, describe all the changes. Interested readers can pursue them, and references, further on the website (www.dsm5.org). The next stage will be that of field trials focusing on the reliability and acceptability of new conditions and new criteria. There is still much to do, and there are likely to be plenty of further changes.

The 11th edition of the World Health Organization's classification of disease is also in preparation, though not yet as advanced. It will be important that the detailed algorithms and multiple categories of the DSM can be transformed into a simpler system for the use of non-psychiatrists. In primary care, for example, the need is for broad categories that will generate appropriate action and suitable referral when needed. We can therefore expect to see some clustering of disorders into superordinate classes. This will need to be based upon clinical value rather than brain physiology or genetic aetiology.

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36

Paediatric Neuropsychological Assessment I: An Assessment Framework

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This chapter sets the framework for the companion chapter of Hohnen and Gilmour (see Chapter 37) by discussing theory and issues in neuropsychological assessment and intervention. A summary of the characteristics and parameters of recent and widely used neuropsychological tests is presented in Chapter 37.

THEORETICAL BACKGROUND

What is neuropsychological assessment?

The purpose of a neuropsychological assessment is to raise hypotheses, which may have a neurobiological basis, to explain functional behaviour and relate this to any underlying neuropathology in order to inform treatment, rehabilitation and education. What it is not, is a reductionist use of test results alone to diagnose neuropathology. Neuropsychological assessment is a complex process involving the application of carefully selected tests in conjunction with comprehensive clinical interviewing and specialized observation, encompassing the child's functioning and their familial and social context. A useful summary of what is involved in terms of collecting information, decision-making in the assessment process and interpretation can be found in Goldstein and McNeil [1].

Assessment may be requested when there is: (i) known underlying neuropathology such as trauma, stroke, infections, neoplasms, disease, hypoxia or iatrogenic causes; (ii) mental health issues, for example attention deficit hyperactivity disorder (ADHD); (iii) congenital or syndromic causes; (iv) systemic and metabolic disorders impacting the central nervous system; (v) unknown neuropathology in a child who is not functioning as expected, particularly when there is unevenness in cognitive development; and (vi) for litigation purposes, specifically following accidents, or a combination of these. A request for assessment with no explanation should occur rarely: if this occurs, it is critical to respond by asking about the concerns driving the referral and the purpose of the assessment. There are times when it is decided that neuropsychological assessment is not relevant or appropriate. Such a decision can be taken at any time during the process. When this occurs, it should be stated clearly, with reasons for the decision given to both referring clinicians and the family.

While psychosocial reasons may explain behaviours in children referred to neuropsychological services, clinicians working with children in mental health should always consider whether behaviour has a possible neuropathological cause. For example, an unreported brain injury acquired a few years ago may explain failure to listen, impulsiveness and forgetfulness, which might otherwise be seen as oppositional behaviour.

The importance of a developmental model

A developmental model characterized by change is needed when assessing children. Neuropsychological functioning changes with age in adults, but change is relatively slow and assessments at ages 25 and 45 may show relatively small

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differences, except in the case of underlying pathology. In contrast, change, often rapid, *is* expected in children. Alteration in the trajectory of previous normal development, or failure to develop between two assessment points is the critical issue. Even a relatively small loss of skills should be taken seriously in children even though this can be a feature of typical development.

Neurological damage may lead to different problems depending on the age at injury, and the age at which assessment is undertaken. For instance, damage to language areas at 3 or 4 years of age may lead to problems coping with complex grammatical structures, but at puberty might result in problems with abstract thought and reasoning. If injury occurs at a time of rapid change, there is an increased likelihood that functional damage will be greater than when development is slow. Knowledge of normal development is therefore an absolute prerequisite in assessment and formulation in child neuropsychology.

Theoretical models of child neuropsychological assessment

Two of the more influential models of child neuropsychological assessment are a functional-organizational approach [2] emphasizing brain–behaviour relationships and relationships between behaviours, and a systemic approach [3] emphasizing the interactive interplay between the neurobiological, psychological and environmental aspects of development. The former model considers three levels of information in assessment: the behavioural symptoms, the associated cognitive profile and the neuropathology, and emphasizes the importance of change. The latter model emphasizes both the neurological and psychological aspects in terms of developmental timing, the context and neuropsychological systems, and particularly three different axes of brain structure: laterality (i.e. left/right), anterior/posterior and cortical/subcortical.

KNOWLEDGE BASE AND COMPETENCIES

Clinicians working in mental health will have competency at carrying out basic tests such as the Wechsler Intelligence Scale for Children (WISC-IV) [4], but will need more specialist competencies to administer and interpret neuropsychological assessments [5], as well as specialist knowledge of neuropsychology. Table 36.1 summarizes both competencies and breadth of knowledge required.

INFORMATION GATHERING

Psychologists carrying out a neuropsychological assessment will themselves need to gather a wide range of information and interview children and families before commencing testing. Reviewing the *medical notes* goes beyond mental health records or family doctor notes, and should include hospital records, where accessible. If questionnaires and rating scales can be completed by families before the young person and family are seen, inconsistencies and concerns can then be investigated in depth during the clinical interview. Time can be saved, which is important in a service where assessments are time-consuming. Throughout the information-gathering process hypotheses can be regularly created and revised, that is after reviewing medical/hospital notes, at the clinical interview, on the basis of test results and observations during assessment, and so on, before a final (working) formulation is reached.

ASSESSMENT ISSUES

As well as the neuropathology, there are other factors that will influence neuropsychological functioning and hence the outcome of the assessment, which need to be considered.

Environmental influences

The social, cultural and ethnic context of development of any child referred to psychological services at any time is important. This is critically so when planning formal testing. Judging the appropriateness of undertaking a psychometric assessment will include whether English is a second language, whether the child has had exposure to different social mores, experiences and educational systems, and general psychosocial influences on development.

Informal observations

General observations of a child during interview and while carrying out tests, the child's strategy, engagement, application and effort can be useful as a means of interpreting results and in final formulation. The validity and reliability of such

Table 36.1 Areas of specialist knowledge and competency required when undertaking neuropsychological assessment.

Area	Breadth/examples
Specialist knowledge	
Fundamentals of neurobiology and development	Neurobiological development; theoretical models of brain/behaviour development, linkages to experiences and atypical environments
Clinical developmental cognitive neuroscience	Basic principles underlying common brain/behaviour techniques, e.g. neuroimaging and electrophysiology
Development of sensory, motor and cognitive neural systems	Neuroanatomy; neural cognitive systems and cognitive trajectories; neural plasticity and reorganization
Developmental disorders, their profiles and functional implications	Neuropsychological systems such as executive and memory systems; and neuropsychological disorders such as epilepsy, hydrocephalus, consequences of abuse and neglect, etc.
Neurodevelopmental assessment in key phases	Infancy, early childhood/preschool, childhood, adolescence
Rehabilitative practice in educational and specialist settings	
Professional issues for paediatric neuropsychologists	Maintenance of competence; working in legal contexts, etc.
Specific competencies	
Test selection for specific neuropsychological problems	Taking account of age, function, test psychometric properties
Test administration and behavioural observations during testing	
Test scoring	
Test analysis and interpretation	Taking account of behaviour during testing, and feedback, including writing both specialist reports and reports comprehensible to carers

microanalysis may be questionable, so hypotheses derived from this informal information need to be treated cautiously and corroborated by interviews and test results. Any observed behaviour may give rise to a number of hypotheses. For example, a child giving short, inadequate answers in the Verbal Comprehension subtest of the WISC IV could have: (i) a hearing problem; (ii) poor attention; (iii) poor language comprehension; (iv) failure to understand the meaning; (v) a memory problem; (vi) speech difficulties; (vii) word finding difficulties; (viii) problems with planning and initiating a response; (ix) anxiety or depression; and (x) oppositional behaviour, or a combination of these. See Table 36.2 for a more detailed description of observations and presenting problems [6].

Setting and task characteristics

When considering children's test results and their behaviour during assessment, it is important to bear in mind the importance of both the characteristics of the testing situation and the task demands [7]. This analysis may explain why some children do well in one-to-one testing, but poorly

Table 36.2 Examples of observations during assessment, by domain.

Domain	Example of observations
Attention	Needing regular refocusing to task Requesting questions/instruction be repeated Early failures but passing more difficult subtest items Slowing down and stopping halfway through subtest items Partial understanding of longer instructions Needs gesture or verbal prompts to scan visual information Fiddles and fidgets
Memory and learning	Forgets questions/tasks Frequent requests for questions/instruction to be repeated Qualitative analysis of how stories/word list are recalled (e.g. correct sequence) Forgets where consulting room is after break Looks for prompts/clues from clinician or parents
Language	Needs simplification of questions/instructions (where possible) Socially quiet Failure to initiate conversation Grammatical and articulation errors Word finding difficulties Simplified or inadequate verbal answers Difficulties making inference or in abstraction Better at leading/controlling than following conversation
Visuoperceptual skills	Writing/drawing untidy, uneven, too large or too small Poor visual scanning strategies Consistently misses information in one part of visual field Better with concrete than abstract visual information Seems unable to 'get the picture'
Executive skills	Poor task planning Needs prompting to initiate tasks Starts impulsively and tries to restart Failure to change strategy despite restarting Perseveration in response to tasks Unaware and/or unconcerned about failure Socially inappropriate/disinhibited Bizarre or unusual answers

in school or at home. The testing situation itself is usually quiet and distraction free, unlike many classrooms, although like many classrooms it is structured and relatively contained. Conversely home may be quieter but freer and less structured. Consequently children, who need help and structure to focus and sustain attention and are easily distracted by what is going on around them, may do better in testing than elsewhere. Others may become anxious in a setting where they become the focus of attention themselves.

Individual tasks in tests may be structured or unstructured, timed or untimed, and require children to work quickly or at their own pace. Open-ended questions or forced choice answers, predictability and prompting are characteristic of some tasks and not others. All this affects an individual child's test performance.

INFORMAL ASSESSMENT

It is important to note that while testing is commonly necessary, neuropsychological assessment can be made without testing, where children cannot or will not be formally tested. It is still possible to gain critical neuropsychological information through talking systematically to them about everyday activities, a process that is in any event helpful prior to testing. For instance, discussion with a 13-year-old, who refused to be tested, about his model-building hobby revealed: (i) his approximate level of reading ability and understanding through his intelligent discussion of the content of an adult magazine on model-building; (ii) his executive skills when talking about how he *initiated* and *planned* building his models, and *organized* the parts and tools he needed before starting, and his *intention* to save future birthday money to purchase further model kits; (iii) his memory and attention through his speaking about visiting an exhibition about model-building, and his ability to focus and concentrate for 2 to 3 hours while he worked; and (iv) his social awareness when asked about issues relating to a model-building club and the model-building community; and the importance of safety when using adhesives and Stanley knives.

CONCLUSIONS: CREATING THE BALANCE

Neuropsychological assessment of children is a specialist, complex process, and it is time-consuming. It can take at least a half-day or commonly longer to get an appropriate history, select, administer and score several tests, complete a clinically relevant report and provide feedback to the child and family. Given the pressures on health-care budgets, the time and other resource costs of such a process are likely to be questioned. While recognizing the importance of such issues, the primary task of the neuropsychologist remains. This is to ensure that the assessment is sufficiently comprehensive and accurate, and that it responds to the needs and concerns of the child, the family, and the requests of the referrers. This will enable the assessor to take account of the strengths and neuropsychological difficulties of the child in planning intervention, education and care.

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37

Paediatric Neuropsychological Assessment II: Domains for Assessment

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INTRODUCTION

We provide here an introduction to the characteristics and parameters of specialist child neuropsychological assessments for clinical or research purposes, as a companion chapter to that of Judith Middleton (see Chapter 36). We discuss measurement issues, the main neuropsychological domains requiring specialized assessment, some relevant standardized assessments appropriate to these domains, alongside a consideration of their psychometric properties.

WHY UNDERTAKE A SPECIALIZED NEUROPSYCHOLOGICAL ASSESSMENT?

For the purposes of this review, specialized tests are those that describe specific aspects of brain function, such as memory and language, as opposed to tests of general ability (IQ). Many specific areas of cognitive functioning contribute to IQ; therefore one would predict that an individual with an IQ below 70, for example (where the mean is 100 and the standard deviation is 15), would score in the low-performance bands of specialized tasks. On the other hand, some individuals, including those with average or high-range IQ scores, have a markedly low test performance in one or more areas of specific brain functioning relative to general ability. Such individuals are described as having a specific learning difficulty (SLD). Specialized neuropsychological assessment is therefore likely to be important in identifying the nature of an SLD for single clinical cases or groups in research.

WHEN IS A SPECIALIZED NEUROPSYCHOLOGICAL ASSESSMENT JUSTIFIED?

Gaining an objective measure of IQ and attainment is usually the first stage of a clinical or research investigation into cognitive functioning, and can be obtained by full administration of an age appropriate standardized IQ assessment, such as the Wechsler Intelligence Scales for Children (WISC-IV^{UK}) [1]. In many cases no further testing is warranted, for example when an IQ test indicates that a child has general learning difficulties. However, there are two common scenarios where additional specialized neuropsychological assessment is justified:

- Groups or individuals with a markedly uneven IQ profile may require an assessment of specific cognitive functioning. Differences in general-ability domains that have statistical significance (i.e. the probability that the difference found between groups could have occurred by chance) are, by definition, relatively common, but discrepancies that have clinical significance (which considers how often this difference would be found in the population) are usually notably larger and may warrant further

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investigation. For example, individuals with a disproportionately low perceptual reasoning (PR) factor score relative to other factor scores (verbal comprehension, working memory and processing speed) on the WISC-IV^{UK} may have visual difficulties, dyspraxia (clumsiness), visual motor integration problems or – since a high PR score depends on a swift response – simply low motivation. Tests of specialized neuropsychological functioning can be used to exclude competing explanatory hypotheses.

- A child who has an attainment level significantly below the predicted level given their measured IQ may have an SLD such as primary language or literacy dysfunction. It is important to note that low attainment relative to IQ is common in children of school age. Many factors could explain this profile, including emotional and behavioural difficulties or school-based variables. In other words, an SLD is a possible cause for poor school performance relative to general ability, but it is not the only feasible explanation.

MEASUREMENT CONSIDERATIONS

A number of issues should be considered when assessing paediatric and clinical populations.

Developmental considerations

Broadly speaking there are relatively few specialized tests appropriate for preschool children. In part this is because very young children are a challenge to test reliably and their neuropsychological function is more difficult to capture. There are a few tests that are useful adjuncts to a general IQ assessment, such as tests of phonological processing [2], working memory [3] and declarative memory [4]. When interpreting data at this young age, bear in mind that some children may 'catch up' in their test performance over time because of neural plasticity or behavioural compensation strategies. For research studies in particular it is often interesting to take a developmental approach in the assessment of specific areas of functioning.

Be sure of why the child fails a task — what is their route to failure?

The non-specific abilities required to complete a given task should be considered, as there are many routes to failure. Many clinical populations have complex neuropsychological cognitive profiles. For example, reading problems are proportionately more common in children with attention deficit hyperactivity disorder (ADHD) [5]. Many, but not all, children with ADHD perform poorly on the Continuous Performance Task (CPT) [6], a test of selective attention. McGee *et al.* report that children with reading disorder score significantly lower than non-reading-disordered groups on the CPT [7]. For such reading-disordered children it would be wrong to conclude that a low score on the CPT necessarily indicates difficulty with the target skill – that is, selective attention. Reading-disordered children have difficulty processing moving visual stimuli [8]. The CPT includes such stimuli but the aim of the task is to capture the ability to attend to pertinent information and screen out irrelevant data, rather than to assess generic visual processing abilities. In other words, the CPT identifies children with target function difficulties (selective attention) and those who have problems with the non-specific demands of the test (processing dynamic visual stimuli).

Where possible, clinical populations should be assessed using a number of tests, presented in a variety of modalities that purport to assess the same target function so that specific deficits can be identified.

Psychometrics

Reliability and validity: Some published tests have questionable psychometric properties. In some cases, the reference populations are inadequate and it is important to look at the 'N's before interpreting scores with confidence. Reliability and validity are also important considerations when choosing a test and deciding how much weight to place on findings. There is no objective cut-off but the general consensus is that, in relation to reliability, a correlation coefficient (r) of greater than 0.6 on test-retest is the accepted minimum for a test to be judged reliable. The validity of a test is measured in a number of ways, the most important of these being construct validity (the degree to which the test measures what it purports to measure).

Interpreting scores: Be careful when interpreting many test items not to overinterpret one or two outliers in the sample of tests as indicating real deficits. There is a risk of making a type 1 error (reporting a difference when there is none). Composite scores are created from individual

subtest scores. These are more reliable than interpretations based on individual scores as the standard error of measurement is reduced due to the larger number of items making up the score [9].

DOMAINS OF SPECIALIST ASSESSMENT

Tables 37.1 to 37.6 review a selection of published tasks assessing specific aspects of brain function. They include target functions and some of the non-specific skills required to complete the task.

General ability

Any specialized neuropsychological assessment relies on measured IQ to establish a general level of cognitive functioning, as an indicator of general ability. A full IQ assessment is usually required, although for research purposes a short version of a test can often be used from which to calculate a pro rata full-scale IQ score. Crawford *et al.* have recently published on the reliability of a short-form administration procedure for the WISC-IV [10].

Memory

Standardized tests in this domain (Table 37.1) assess explicit memory or conscious recollection (for facts or events) as opposed to implicit (for skills or procedures) traces. There are separate dimensions of memory – working (short-term), stored (long-term), verbal, spatial (visual) and learning capacity. Individuals may have impairment in one domain but not in another. In addition, it is important to test both delayed recall and recognition. Children who do poorly on a test of recall but accurately recognize previously presented items can often store information but have problems accessing it.

Language

Tests of language assessment fall into two categories: receptive and expressive. Visual language channels are independent from spoken language channels, and so assessments that focus on spoken language (reviewed in Table 37.2) do not necessarily exclude written language problems. However, specific written language impairment and spoken language vulnerabilities often co-occur [11]. Assessments of written language are likely to be classed as assessments of attainment, such as subtests of the Wechsler Individual Attainment Test [12].

Attention

Attention has two main components: sustained (effortful processing over a significant period of time) and selective (vigilance for target stimuli while ignoring distracter stimuli). Many children with ADHD do poorly on these tests but there is no diagnostic cognitive test for the condition. It is identified on the basis of a pervasive behavioural profile rather than performance on a cognitive task. Until recently, many tests of attention for children were rather theoretical, attempting to define a core cognitive deficit in children who have the ADHD behavioural profile (the debate about the existence and nature of such a core deficit continues). The Test of Everyday Attention for Children (TEACh) [13] provides a battery of tests of attention and inhibition presented in a variety of visual and auditory modalities (see Table 37.3).

Spatial ability

Spatial skills include the ability to mentally rotate visual configurations in space and to recognize that same configuration, regardless of its orientation. The Benton Face Recognition Test [14] (see Table 37.4) is a good example of a visual orientation task that uses meaningful stimuli. The Mental Rotation subtest of the British Ability Scales [15] – note that this is not the most recent version – assesses orientation using abstract stimuli.

Spatial ability also includes the naming of objects – though it could be argued that naming makes such high demands on visual memory that it is better described as a visual memory skill rather than a spatial ability per se. The Gestalt Closure subtest of the Kaufman Assessment Battery for Children is a test of visual naming that is appropriate for children [16].

Motor skills

Motor tests (Table 37.5) assess a number of separate elements – strength, speed and dexterity. Many tests of motor dexterity include a visual component (e.g. the Rey–Osterrieth Complex Figure) [17,18]. The Visual Motor Integration Test is a useful tool to assess visual, motor and visuo-motor integration skills [19].

Executive function

Executive function (EF) includes initiation, planning, inhibition, flexibility, self-regulation, concept

generation and working memory (Table 37.6). It is argued that grouping these together into a unitary concept is flawed as they are so diverse. There is also controversy over the construct validity of the tasks that profess to assess EF. There is strong clinical and theoretical justification to develop more refined classifications of the functions associated with EF, particularly as investigations of EF are often central to the assessment of many clinical conditions. Deficits in EF are implicated in many disorders (e.g. ADHD, autistic spectrum disorders and schizophrenia) [20–22]. In addition to the psychometric assessments of EF outlined in Table 37.6, the Behavioural Rating Index of Executive Function (BRIEF) [23,24], a parent, teacher and child rating questionnaire, provides information about behaviours that are associated with executive function difficulties (preschool and school age versions).

Social cognition

Social cognition (see Table 37.6) covers many high-order brain functions, such as the expression and understanding of emotion, facial expression and subtleties of language embedded in social interaction. 'Theory of mind' describes the ability to 'mentalize' and infer another person's state of mind. For the purposes of this review, theory of mind is not considered an aspect of social cognition, but is regarded as a theoretical concept. Describing theory of mind as a concept rather than a brain function is not an attempt to disregard the significant empirical data showing that children with autistic spectrum disorders, particularly low-functioning individuals, perform poorly on theory-of-mind tasks.

The measurement of social cognition relies on consensus opinion, in contrast to other brain functions, which can be quantified using objective right or wrong answers. The complex nature of social cognition may explain why there are few standardized tests assessing this aspect of functioning, but the paucity of standardized measurement is a challenge to those working in the specialist assessment of cognitive functions. The NEPSY-II (2007) has two subtests that measure 'Theory of Mind' (ability to understand mental functions such as belief, intention, deception, emotion, imagination and pretending as well as how emotion relates to social context) and 'Affect Recognition' (ability to recognize affect) [25].

CONCLUSIONS

This chapter outlines measurement and testing considerations in the field of paediatric neuropsychology. As a clinician or researcher one must consider how to interpret test results in the context of neuropsychological theory, and one should also include considered dissemination of test results. Empirical data ideally indicate specific recommendations, which may simply raise awareness about a child's profile to the child themselves, family, school staff and other professionals so that the environment may adapt to accommodate a child's cognitive vulnerability. In some cases, test results may indicate an intervention that could improve cognitive functioning in the child. These principles of careful interpretation apply equally to clinical cases and research populations. In some instances, results could inform theory – for example, group data could point towards a function (or particular phenotype) and may be mapped genotypically, or results could be correlated with neuroimaging data. Our task as professionals might be summarized as raising awareness about the utility and delicacy of paediatric neuropsychology.

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Table 37.1 Memory assessment measures.

Assessment	Target function	Reference population (*n*) in each age band	Non-specific abilities*	Age range (to nearest whole year)	Comments
Automated Working Memory Assessment [26]	Memory — short-term and working memory Visual-spatial and verbal	59—67		4—22	Computer administration and scoring
Working Memory Test Battery for Children [3]	Memory — short-term and working memory Visual-spatial and verbal	59—100		4.7—16	
Children's Auditory Verbal Learning Test — 2 [27]	Memory — verbal, immediate and delayed recall and recognition	30—81		6—17	
Wide Range Assessment of Memory and Learning [28]	Memory — verbal and visual, delayed and immediate, recall and recognition	110—117		5—15	
Rey—Osterrieth Complex Figure [29,30]	Memory — visual, immediate, delayed and recognition	18—48	Planning, visuo-motor skills	6—15	
Children's Memory Scale [31]	Memory — visual, verbal, immediate, delayed recall, attention, recognition, learning	100		2—16	It is possible to predict a General Memory Index Score from WISC FS IQ
Digit Span (WISC-IV^{UK}) [1]	Memory — auditory working	74		6—16	
Rivermead Behavioural Memory Test [32,33]	Memory — everyday tasks	100		5—14	
The Visual Memory Battery [34]	Memory — working and stored, recognition and learning	40	Sustained attention (Matching to Sample subtest)	4—adult	Computer administration and scoring. Motor speed is controlled
NEPSY — memory subtests [25]	Memory — visual, verbal, immediate and delayed	100		3—16	Appropriate subtests: names and faces; narrative; sentences; list learning
Color Object Assessment Test (COAT) [4]	Declarative memory	94—139 per 6-month age band		18—36 months	Only preschool memory assessment

*Intact senses and motivation are assumed in all cases.

Table 37.2 Language assessment measures.

Assessment	Target function	Reference population (*n*) in each age band	Non-specific abilities*	Age range (to nearest whole year)	Comments
Clinical Evaluation of Language Fundamentals — 3 [35]	Language — spoken expressive and receptive	151—267	Auditory attention	5—adult	Test—retest reliability on some subtests is low
Clinical Evaluation of Language Fundamentals — preschool [36]	Language — spoken expressive and receptive	100	Auditory attention	3—6	Test—retest reliability on some subtests is low
Test for the Reception of Grammar (TROG) [37]	Language — spoken receptive grammar	120—217		4—adult	TROG-E was published in 2005 and is a computerized version of same test
British Picture Vocabulary Scale [38]	Language — spoken receptive naming grammar	183—423	Visual discrimination	3—16	1997 stimuli are less ambiguous than those in previous editions
Renfrew Language Scales [39]	Language — spoken comprehension, word finding, expression, narrative speech	58—101		3—8	
Token Test [40]	Language — spoken receptive comprehension of language concepts	29—53	Short-term (working) auditory memory	6—13	
NEPSY — language subtests [25]	Language — expressive and receptive, cognitive processes related to language	100		3—16	Appropriate subtests: phonological processing; speeded naming; repetition of non-words; comprehension of instructions

*Intact senses and motivation are assumed in all cases.

Table 37.3 Attention assessment measures.

Assessment	Target function	Reference population (*n*) in each age band	Non-specific abilities*	Age range (to nearest whole year)	Comments
Continuous Performance Test [6]	Attention – visual sustained attention and impulsivity (behavioural inhibition)	40	Age-appropriate reading	6–adult	Gender-differentiated norms
Test of Everyday Attention [13]	Attention – auditory and visual sustained and selective attention, response inhibition	29–58	Basic numeracy is required for some subtests	6–15	
Cambridge Neuropsychological Test Automated Battery [34]	Attention – sustained, selective and divided	40		4–adult	Computer administration and scoring. Motor speed is controlled
NEPSY – attention subtests [25]	Attention – auditory – selective and sustained (vigilance)	100		5–-16	Appropriate subtest: Auditory Attention and Response Set

*Intact senses and motivation are assumed in all cases.

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Table 37.4 Spatial/visual assessment measures.

Assessment	Target function	Reference population (*n*) in each age band	Non-specific abilities*	Age range (to nearest whole year)	Comments
Developmental Test of Visual Motor Integration [19]	Visual discrimination, motor skill and visuo-motor integration	6—16	Impulsivity may interfere with performance in the motor skill subtest	2—14	Some debate regarding the graduation of test item difficulty
Trail Making A and B [41—43]	Visual search and sequencing/motor output	10—101	Knowledge of number and alphabet sequence	6—15	Parts A and B measure different functions
Mental Rotation — British Ability Scales [15]	Visual — rotation of abstract figures	90—189	Conceptual ability to take another person's perspective	8—14	
Gestalt Closure — Kaufman Assessment Battery for Children [16]	Visual — meaningful stimuli naming	200—300	Knowledge of industrialized world objects	2—13	
Face Recognition Test [14]	Visual/spatial ability — face recognition	19—59		6—14	
Judgement of Line Orientation [44]	Visual—spatial judgement	23—50		7—14	
Rey—Osterrieth Complex Figure Test (copy condition) [17,18,29,30]	Visual/motor planning	18—48		6—15	
Right-left orientation [45]	Spatial discrimination	7—16		6—16†	
NEPSY — visual spatial subtests [25]	Motor and visual perception; line discrimination	100		3—16	Appropriate subtests: Design copy; arrows; route finding

*Intact senses and motivation are assumed in all cases.
†Some extrapolated norms.

Table 37.5 Motor assessment measures.

Assessment	Target function	Reference population (*n*) in each age band	Age range (to nearest whole year)	Comments
Finger Tapping Test [46]	Motor speed	20	6—14	Gender-differentiated norms. Boys are significantly better at this task
Purdue Pegboard Test [47,48]	Motor dexterity (fine)	23—40	5—15	Practice effects are notable
Cambridge Neuropsychological Test Automated Battery [34]	Motor speed and reaction time	40	4—adult	Computer administration and scoring. Motor speed is controlled
Grip strength [46]	Motor strength	20	6—14 (no norms for 9—11)	Sex and hand preference differentiated norms
NEPSY — sensorimotor subtests [25]	Motor dexterity and motor speed; imitation of sequences; graphomotor speed and accuracy	100	3—16	Appropriate subtests: fingertip tapping; imitating hand positions, manual motor sequences, visuomotor precision

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Table 37.6 Executive function and social cognition assessment measures.

Assessment	Target function	Reference population (*n*) in each age band	Non-specific abilities*	Age range (to nearest whole year)	Comments
Delis–Kaplan Executive Function System (D-KEFS) [49]	Executive function	Approx. 100		8–adult	Some parallel versions of tasks. Good variety of tasks
Behavioural Assessment of Dysexecutive Syndrome (BADS) [50]	Executive function – predicts everyday function	22–32		7–15	Computer administration and scoring. Motor speed is controlled
Cambridge Neuropsychological Test Automated Battery [34]	Executive function – working memory and planning	40	Sustained attention (matching to sample subtest)	4–adult	Computer administration and scoring. Motor speed is controlled
Trail Making A and B [41,42,43]	Executive function – motor planning and disinhibition	10–101	Number and alphabet sequence ability	6–15 (some extrapolated norms)	Parts A and B measure independent functions
Rey–Osterrieth Complex Figure Test (copy condition only) [17,18,29,30]	Executive function–visual planning	18–48	Visuo-motor skills	6–15	
Wisconsin Card Sorting Test [51]	Executive function – cognitive flexibility; concept formation	27–55	Colour vision, basic numeracy	6–adult	There is a positive relationship between years in education and performance
Stroop Word and Colour Test [52,53]	Executive function – inhibition of a prepotent response	14–29	Colour vision, literacy	Collated norms for 7–16	All three conditions must be administered to control for speed of processing
Diagnostic Analysis of Non-verbal Accuracy 2 [54]	Social cognition – receptive non-verbal ability; voice and face recognition	25–305	Sustained auditory attention	3–adult (collated norms)†	Body language subtest has been dropped for the most recent edition
NEPSY – attention and executive function subtests [25]	Executive function – inhibit automatic response, planning and organization, shift set	100		3–16	Appropriate subtests: inhibition, clocks, animal sorting, design fluency, Statue

*Intact senses and motivation are assumed in all cases.

†Child faces only.

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Assessment of Child Psychiatric Disorders

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INTRODUCTION: WHY DO A PSYCHIATRIC ASSESSMENT?

What is the rationale for choosing to undertake a psychiatric assessment? Various answers to this question have been suggested. For instance, Harrington [1,2] argues that a psychiatric assessment focuses on four key questions:

- is a psychiatric disorder present?
- what sort of disorder is it?
- what other problems are present?
- why does the patient have the presenting problem(s)?

Hoyos [3] takes a more developmental perspective and contrasts assessment in child psychiatry with assessment of psychiatric disorders in adults, arguing that understanding child development is critical when assessing children as normative behaviour changes with developmental age. Scott [4] argues that making a correct diagnosis requires applying a body of knowledge based on extensive research. The American Academy of Child and Adolescent Psychiatry (AACAP) Practice Parameters and Guidelines [5] say that the psychiatrist needs to prioritize symptoms and diagnoses so that a reasonable treatment plan can address multiple problems.

As we illustrate below, all of these rationales emphasize important aspects of the psychiatric diagnostic process. In a busy, multidisciplinary, generic child mental health service, an additional question might be posed concerning which children need to be assessed by a child psychiatrist. Given the pragmatics of time and availability, when is it important to apply the specific additional skills utilized in psychiatric diagnoses? We would suggest that psychiatric assessments be undertaken when the following questions are pertinent:

1. Is there a coexisting significant medical problem where medical skills can contribute to understanding the illness process?
2. Is there a question of diagnosis – for example, whether the child's symptoms meet the diagnostic criteria for a bipolar disorder?
3. Is there a likelihood that medication will be required as part of the management/treatment plan?
4. Is an admission to hospital for further assessment or treatment required?

SETTING THE STAGE FOR ASSESSMENT

The general principles of assessment outlined apply to children of any age. However, it is particularly important to bear in mind that the range of psychiatric disorders seen may differ according to the child's age and developmental stage.

The assessment of a child and adolescent usually will take place over several meetings, using information from several different sources. An initial assessment interview is a key time to begin the therapeutic process of engagement with a family and it is important to try to get it right. It is also a key part of any assessment of factors that may pose a risk to the child's safety or well-being.

Consideration should be given as to whom to see. With a younger child, it is usually helpful to

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meet with the whole family at the start of the interview, but at some stage in the assessment of a younger child, the parent/s or guardian should be seen without the child being present. Where it is age appropriate, a younger child must also be given time to speak with the interviewers – either by focusing some of the family interview on them, or by meeting with the child alone. An adolescent will usually wish to be seen for some of the assessment without their parents. The assessment interview unfolds in a number of stages that are outlined below.

INTERVIEWING FAMILIES

Family interviews should begin by making the family welcome and putting them at their ease. Then, exploration around key issues follows: what do the family see as the problem, and who has the problem? How are they trying to deal with it at present? What have they already tried and what help have they already received? How does it affect their lives?

One strategy the assessor may use is to ask the family to work together in the session on an exercise, for example drawing up a family tree. This gives the assessor an insight into how family members relate to each other and also into the presenting problem. Are the parents sensitive to the child's communications? How do they respond? What is the parent's own relationship like? Is one family member ignored?

It is also important to ask who else is important to the child but is not present. This could be a separated parent, or a grandparent, for example. How would the family describe this absent person, and what would they say if they were in the room?

INTERVIEWING CHILDREN AND ADOLESCENTS

With the younger child, it may not be possible formally to examine the child's mental state and the clinician will need to rely on observation. This should include an observation of the child's behaviour, interactions with family, interactions with clinicians, and play. Key aspects of the child's functioning and behaviour to note are shown in Table 38.1.

It is important to take into account developmental issues and have a good understanding of

Table 38.1 Observing the child. Adapted from Bruce and Evans [6] and Bruce and Keene [7], with permission.

Aspect	Observations
Behaviour	Appearance and dress
	Emotional responsiveness
	Mannerisms and stereotypies
	Activity level
	Any risky behaviour
Talk	Form:
	• coherence and speed
	• spontaneity
	• spontaneity
	• prosody
	• articulation
	Content:
	• persistence
	• interruption of attention
	• child's interests
Anxiety and mood	Fearful or anxious
	Restless
	Disturbed or aggressive
	Withdrawn or shy
	Sadness
Interactions	Parent—child interaction
	Interaction with other family members
	Interaction with clinician
	Interaction with the environment — toys and play
Intellectual functioning	A brief overview of the child's current level of cognitive ability will be needed
	Does this differ greatly from their chronological age?

the particular young person's stage of emotional, cognitive and psychological development, which may differ according to their physical maturity. It is important that the child's or adolescent's individual autonomy is given due respect. This issue can cause tension in the assessment phase, but should not be ignored. Confidentiality is linked with this issue. It will be important that children can speak in confidence about certain issues. However, the limits to confidentiality with respect to concerns about the safety/well-being of themselves and others need to be outlined to them. Thus, it may not always be possible to guarantee full confidentiality, and the young person is entitled to a full explanation of how this right will, as far as possible, be respected. Many of those young people whose difficulties persist into adulthood will have their first encounter with mental health services during their childhood. The nature of this experience will affect their future compliance and engagement with mental health services, so it is especially important to consider the long-term implications of their interaction with professionals at this stage.

When seeing an adolescent it is helpful to establish what they personally see as the problem. They may not agree they have a problem at all. Even if they do not agree with the parent or referrer, the examiner can still establish a rapport, and it is important to engage with the young person. As with younger children, the issue of confidentiality and its limits should be established. The process of assessment should be explained clearly. A psychiatric mental state examination should be carried out (see Box 38.1). The interview should also include a sexual, forensic, and drug and alcohol history.

It is important to end the interview in a way that leaves children and adolescents feeling that their perspective matters. Other useful information can also be gained at this stage. It is important to ask the young person if there is anything else they would like to talk about or that they think the interviewer should know that would be helpful for the assessment. It is also important to ask if they want to ask any questions about the assessment, possible interventions, or any other matters of concern or interest to them. With a younger child, a helpful technique is to ask the child: 'if you had three wishes what would you wish for?' They often respond giving useful information about their

Box 38.1 Mental State Examination (MSE)

An MSE should be conducted with every child and adolescent being assessed. It is informed by the child's history, the assessment of other relevant factors and the conditions under which the assessment is taking place. It will also be informed by the developmental stage of the child/adolescent.

The MSE is used as part of the wider formulation to plan the next steps in management. It is an active process conducted as often as needed throughout any intervention with the child/adolescent.

The domains covered in a MSE include:

- Appearance, attitude and behaviour
- Mood and affect
- Speech
- Thought processes and content
- Perception
- Cognition
- Insight and judgement
- Risk

Clinicians will need to draw on their communicative skills with children and adolescents in order to conduct a successful MSE.

situation, fears and worries, using terms and a context that is meaningful to them.

INTERVIEW WITH THE PARENT(S) OR CARER

It is important to obtain from the parent(s) a thorough description, both of the current problem and an account of the child's developmental, medical and school history. The key points are summarized in Box 38.2 [6,7].

The parents can be interviewed together with the young person or separately. Many young people want to know what is being said about them and should not be excluded. At times it may be more appropriate for the parents to be seen separately, as when the young person is extremely agitated or unwell, or if the parents feel concerned about talking in front of the young person. If this is done, the young person should be informed that the discussion is taking place and it should be made clear to all involved what level of information will be shared between family members. It is important to be aware of not being drawn in to colluding with any secrets between family members.

PHYSICAL EXAMINATION AND INVESTIGATIONS

Most children who attend child mental health services are not routinely examined medically. It is important that consideration is given to a physical examination, if one has not already been done by the GP or Community Paediatrician. Reasons for this assessment include the possibility of an underlying undetected physical condition that has caused a child's difficulties, or the prescription of medication that could have physical side effects, such as risperidone or methylphenidate. Guidelines can be found that are relevant to each medication and indicate the essential elements of physical examination – for example, the European Clinical Guidelines for Hyperkinetic Disorder [8] – but most National Health Service Trusts also have their

Box 38.2 Information to be obtained from the parent(s)

- Current problem as parent/s sees it and its effect on the family
- Behavioural problems (e.g. aggression, conduct problems, truancy, running away)
- Any risky behaviour
- Emotional symptoms (anxiety, fears, depression, suicidality)
- Attention and concentration
- Activity levels
- School history, performance and attendance
- Family life and relationships, including any family history of illness
- Peer and sibling relationships
- Any recent adversity (e.g. bereavement, trauma)
- Developmental history
- Physical health
- The child's temperamental characteristics
- The child's strengths
- What the parents see as the child's difficulties
- What help/interventions have already been tried
- What has previously been successful.

Adapted from Bruce and Evans [6] and Bruce and Keene [7], with permission.

own prescribing guidelines that state minimum levels of physical investigations required in their policies. The AACAP Parameters [5] also recommend physical examination or referral to a practitioner who can undertake this. A baseline height and weight should always be recorded. Where there is suspicion of an underlying medical condition, a full physical examination is essential. A full neurological examination may also be required if there is history indicative of a neurological disorder such as developmental delay, epileptic fits or loss of skills. Other indicators include a history or physiognomy that suggests the child could have a congenital syndrome. Useful pointers to congenital syndromes include learning difficulty, dysmorphic features (including unusual facial features) and extreme values for height, weight or head circumference [1].

Most children will not require further medical investigations unless there is a clinical indication or abnormalities have been found on the physical examination. However, it is important to carry out appropriate blood tests if medication is being considered.

OTHER SOURCES OF INFORMATION

Other agencies, such as education or social services, which are involved with the family or child, may have useful information to help understand the child's problems. Schools can provide particularly valuable accounts of a child's difficulties. It is usually best to obtain structured reports, in the form of a set of specific enquiries. Potential informants include the child's class teacher and any schools special education needs advisors. Depending on the nature of the child's problem, the enquiry can be supported by standardized questionnaires that are available for teachers such as the Conners' Rating Scales for Hyperactivity/Inattention. A school or nursery observation by the clinician (if resources allow) will also yield valuable information. It is important to ensure that parental consent has been obtained if the clinician wishes to seek information from such other sources.

PUTTING IT ALL TOGETHER: CONSTRUCTING A FORMULATION

First of all, the clinician will need to consider whether the child's behaviour, emotional state or presenting difficulty is *abnormal* in relation to his or her age, gender, developmental stage and cultural background. The symptom needs to be persistent, severe and frequent and of a sufficient extent in order to be considered abnormal.

It will also be important to know if the symptom is leading to *functional impairment* in the everyday life of the child. Four main criteria can be used to assess impairment:

1. interference with a child's development;
2. social restriction;
3. suffering or distress to the child;
4. effect on others.

Different aspects of the child's problems can be considered using a multiaxial framework, as has previously been done in the *ICD-10 International Classification of Mental and Behavioural Disorders in Children and Adolescents* (ICD-10). However, revisions to our current classification systems in both the UK and USA are underway, and we refer the reader to Chapter 35.

An adequate formulation of the child's difficulties will require the clinician to piece together the presenting features of the problem, with any aetiological factors, and to comment on the differential diagnosis, management and prognosis. This evaluation will form the basis on which any intervention is planned.

Attention must be paid to risk assessment and associated management, especially with adolescents. Risks can include risk to self, risk to others (within or outside of the family) or risk of abuse/neglect. The psychiatrist plays a crucial role in risk management, and should attempt to manage anxiety within the young person's social network.

Formulation also requires the assessor to have a good understanding of strengths, resilience and protective factors. These can be exploited in the management plan and enhance outcome.

ACKNOWLEDGEMENTS

We acknowledge with thanks Quay Books, Mark Allen Healthcare Ltd, for giving permission to use Table 38.1 and Box 38.2 as updated and adapted versions from Bruce and Keene [7].

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39

Psychological Assessment

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Psychological problems in children and young people commonly result from the interplay of a complex of factors over time. The same problem shown by different individuals can arise for any of a variety of reasons, and the same instigator can lead to individuals showing different problems. Aggression at home may occur because one individual is socially isolated, another is not coping with the learning demands of school, or in a third, because of family tensions. One child bullied at school may become withdrawn and uncommunicative whereas another may be aggressive at home and disruptive in school. Complexity is increased because the factors that give rise to difficulties may not always be the same as those maintaining them. Rejection by peers can lead to a loss of self-esteem, and even if subsequently reaccepted, the individual may remain hypersensitive to signs of rejection.

Assessment (or 'evaluation', 'diagnosis', or 'case conceptualization') is the *process* whereby clinicians gather and interpret information from a variety of sources to help understand and manage such problems. The information is elicited and collected using formal (structured) or informal observations and interviews, and special tests. An 'assessment', 'evaluation' or similar refers to a set of statements conveying the clinician's understanding of the nature of the problems and the actions needed to manage them. This *product* of the assessment process, referred to here as a 'formulation', is essentially an explanation or hypothesis about the nature of the problems and what is required to manage or resolve them.

Assessment requires specialist knowledge and skills and is arguably the most critical part of clinical practice: what the clinician learns through their investigations profoundly influences what happens by way of further action. Where the primary focus is on psychological functioning, a major feature differentiating forms of clinical practice is the clinician's theoretical orientation or 'model'. Models influence how the practitioner approaches assessment, how the problems are understood, the nature of the eventual formulations they offer, the interventions undertaken and even the structure of services [1]. This chapter describes an approach to the psychological assessment of children, young people (hereafter children), their families and others involved in their care based on a model derived from several sources.

MODELS

Models in health care commonly reflect the characteristics of medical diagnostic systems for identifying disorders. In simplified form, these view a psychiatric disorder as something in the person caused by a (usually) diagnosable disease or injury, requiring individual treatment by professionals to restore a healthy state. The appropriateness of such models for understanding psychological problems is questioned, reflecting disagreements with both the specifics of diagnostic systems – such as categorical conceptualizations [2] – and the associated approaches to psychological problems [1]. A 'social' model, also simplified, views dysfunction and disability as social constructs, not individual attributes, requiring social change to remove the disabling consequences, ignoring the contribution of biological and psychological factors. From

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a psychological perspective, a model incorporating biological, psychological and social parameters and their impacts on functioning – a *biopsychosocial* model – has greater 'real-world' meaningfulness [3].

A BIOPSYCHOSOCIAL MODEL

A biopsychosocial model recognizes the interplay of multiple factors in the origins, maintenance and impacts of psychological problems on personal functioning. As an alternative starting point, it is complemented by adopting experimental psychology and psychopathology as the primary knowledge bases. For child psychologists, systemic, developmental, individual difference and sociocultural considerations are also basic to their approach. This recognizes that biological systems develop and function in ongoing transactions between genetic endowment and experiences, within and across all levels of the developing organism and environment, from cellular biochemistry to the physical and psychosocial worlds. These influences operate from conception (and before), over the lifespan [4], and may need to occur in certain developmental (sensitive) phases for emerging patterns to function effectively [5]. Postnatal development is recognized as non-linear, involving major qualitative and quantitative transitions from birth onwards. Underlying development are differential changes in neural systems functioning in typical environments. Some systems only become fully functional well into adolescence or beyond [6]. Such requirements for an acceptable model are encompassed in Bronfenbrenner's developmental bioecological theory [7].

In a bioecological-inspired 'model' (see Figure 39.1), the individual is at the centre of interacting nested systems extending from care at home to the broader sociocultural and physical milieu. These influence and can be influenced by the developing individual, 'the biopsychosocial person' at the centre of the microsystem (Ref. [7], p. xvi), and by each other. Bronfenbrenner's [8] ideas have had a profound effect on thinking in developmental psychology, manifest in varied applications of bioecological theory. Figure 39.1 is an instance applied both to understanding clinical problems and to assessment. In this model, the individual is at the centre of increasingly broader

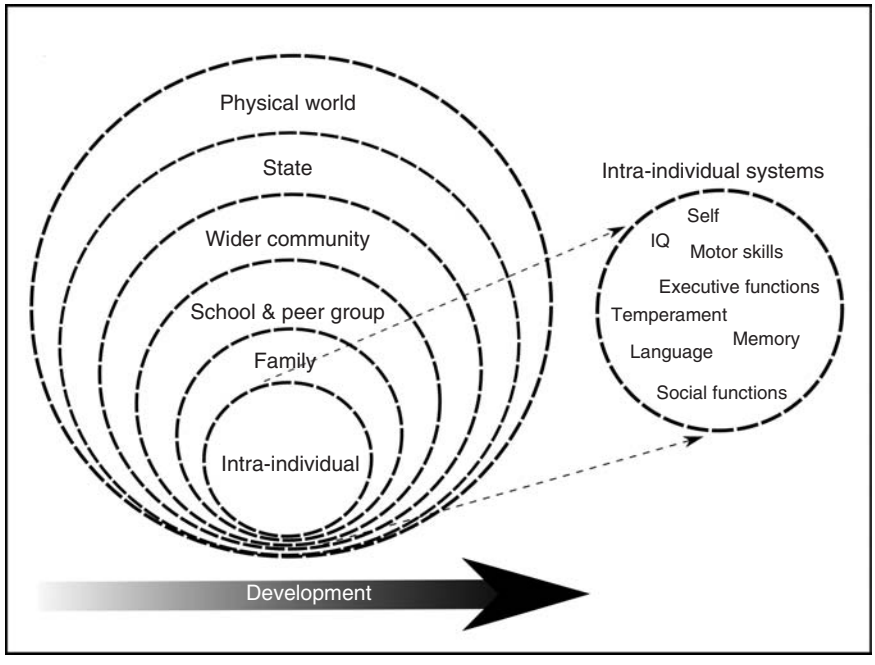

Figure 39.1 Schematic model of the main systems conceptualized as the major components of a biopsychosocial model.

ecologies. The dashed circles symbolize 'gated' bidirectional transactions among and across all the main systems. That is, systems are not necessarily open to all influences: for example, some individuals are more resilient than others; auditory signals inaudible to people influence animals. Within-system transactions occur and impact on other systems. When parents collaborate, rather than argue about the management of their child, this can help the child to feel more secure and confident at home, supporting the parents' engagement. It also leads to the child being more likely to respond positively to their teachers and other adults outside the home.

The intra-individual elaboration on the right of Figure 39.1 illustrates some of the main general within-individual functional areas. These can be differentiated if necessary, for instance, into finer-grain temperamental characteristics [9]. The model is superimposed on a developmental trajectory symbolizing changes over time. The shading indicates that factors operating pre-conception (such as radiation exposure), can influence physical status and functional development of children conceived many years later.

A model such as this has several advantages for thinking about clinical problems and assessment. It identifies potentially influential systems interacting and giving rise to complexity in clinical phenomenology. It provides a structure for systematic assessment, reminding clinicians of the factors within the individual and the broader ecologies that need to be considered. Assessment also needs to take account of typical developmental changes, individual differences, and the ways in which characteristics are manifest in cultural and ethnic variants.

Assessment is commonly structured using a mental or other aide memoir, identifying the domains to investigate that are relevant to the identified problems. Within each, certain features may trigger more detailed probing, including the use of psychological tests. Other areas might be ignored. Investigations can entail family meetings, home or school visits and the like, where information from these contexts might be useful. Importantly, the assessment process should also identify the strengths within the individual and the broader environments that protect and support daily functioning. Such information aids understanding of the presenting difficulties and points to potential resources for intervention. The proposed model thus emphasizes the need to adopt a multilevel perspective in assessment while leaving the clinician free to decide when, where and what to explore in more detail.

Nevertheless, all practitioners, irrespective of approach, must be familiar with and take account of psychiatric and other relevant medical diagnoses and treatments, partly for ethical reasons, partly for risk management, and because the functional impacts found in individuals with such diagnoses commonly need to be managed. For example, an individual with attention deficit hyperactivity disorder (ADHD) whose activity levels and concentration have benefited from medication may still need psychological assessment and interventions to manage the personal and other impacts on family life, education and peer relationships, of growing up with ADHD characteristics.

Ultimately, as with other models, it is the clinician who has to integrate the assessment information into a coherent formulation with implications for intervention. Formulations are tentative explanations of the clinical phenomena. They can change following feedback from clinical encounters, from further investigations, and by the ways in which the individual or family responds. Depending on the assessment, the formulation may lead to one or more individualized interventions, ranging from focal to broad-based, implemented within any or several of the environments where there are significant functional impacts. This approach again stems from recognizing that multiple differing influences may be involved in instigating and maintaining clinical problems. Whether or not evidence-based interventions are used will depend on the availability of suitable procedures, taking account of developmental status and the circumstances of the child or young person.

Although there are differing views about the contribution of psychological tests, when properly used, tests can provide essential information to aid clinical understanding and decision-making, and can be an important source of outcome-monitoring data.

PSYCHOLOGICAL TESTING

A psychological (psychometric) test is a systematic or rule-governed procedure for sampling psychological attributes and processes. These

are not directly observable but are inferred from behaviour that occurs naturally or that is specifically provoked by the test procedure. For instance, written responses to focused questions can tap attitudes to the 'self'; special puzzle tasks require the use of spatial abilities. One of the strengths of psychological tests is that questions and tasks are clearly described in a test manual with everyone asked to do the same thing under similar conditions. Answers or performance on a task are converted to a numerical score, which is compared with the scores of a reference group. This gives an indication of severity, for example of depressed mood, the level of an ability (e.g. above or below average spatial skills) or the extent of functional difficulties such as social skills difficulties.

Clinical tests take many forms, including some that can be administered and scored by a stand-alone computer or over the internet. A number produce computer-generated reports. All tests used clinically should meet accepted quality standards [10], including applicability to the individual to be tested. While tests are relatively easy to administer and score, clinical testing requires advanced knowledge and skills: an understanding of measurement errors and their effects on score interpretation; judgement of the reliability of the results from the individual administration; cross-checking (do the scores make sense?); and incorporating the test results in a meaningful form in the broader assessment. Criteria for ensuring the competence of test users are available [11].

Tests encompass the full range of clinical diagnoses in children and young people [12] as well as abilities, attainments (achievement), temperament, social skills, self-image and other psychological phenomena [13]. Tests of attention, memory and other special procedures, used for instance in neuropsychological assessment, are considered in Chapter 37.

CONCLUDING COMMENTS

This account does not cover the training and experience necessary for the clinical psychological assessment of children and young people, nor the prioritization of objectives and other aspects of the assessment process, including linking it to intervention. Nor does it address applications in stepped and managed care or in other services responsive to the economic and other constraints of health-care delivery. However, the model outlined here provides an adaptable and appropriate framework for thinking about the often complex psychological problems, circumstances and resources of children and young people, and for the clinical assessment and management of such problems.

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40

Family Therapy Assessment

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In this chapter I provide a brief overview of family therapy assessment, outlining how theoretical orientations can influence assessment, the main goals for the assessment process, and the strengths and limitations of a family systems therapy approach to assessment.

WHAT IS FAMILY THERAPY?

Family therapy, also referred to as couple and family therapy, and family systems therapy, is an evidenced-based psychotherapy that seeks to address a variety of emotional, behavioural and other biopsychosocial difficulties through working with family members who are in intimate relationships. Such relationships could include, for instance, married couples, children with their parents, grandparents with their adult children and grandchildren, and other kinship groups. Within modern family therapy, however, all family members do not need to be directly involved in therapy, though their roles and perspectives can be discussed and considered by the family members who are present

From the perspective of the family therapist, the family milieu will have a significant bearing either on the development of a person's presenting emotional and behavioural difficulty and/or on its maintenance. The individual's ability to accommodate and adjust to life experiences such as a change in family configuration or a physical health difficulty, as well as the meaning ascribed to those experiences, will be influenced by other family members.

Presenting difficulties are not, therefore, seen to reside in individuals per se, but to result from a complex interplay of family relationships and processes. For instance, a child's school refusal could be regarded as serving a 'positive' function within the family if it facilitated communications between parents who were otherwise emotionally distant from each other. Equally, the presenting difficulty may have arisen as a result of a family's inability to adapt and adjust to changing family circumstances – as, for instance, when a father who previously worked away from home, now works locally and begins to play a greater role in disciplining the children within the family, a role previously undertaken by the children's mother.

Whatever the family conceives as the 'difficulty' – that is, the reason they are seeking therapy – the family therapist will seek to reduce any associated stigmatization and to work with the family towards a situation where there is some recognition that all family members have an important part to play in facilitating an understanding of the problem and in generating a solution.

Family therapy and family-based approaches have been shown to be effective for a variety of clinical problems, such as anxiety, depression, psychosomatic problems and eating disorders [1]. More detail of family therapy interventions can be found in the Chapter 44.

TYPES OF FAMILY THERAPY AND THE FOCUS OF ASSESSMENT

Over the last 10 years there has been a growth of family therapy models, with each having different theoretical underpinnings, and a variety of psychotherapeutic influences have become integrated

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alongside structural, strategic communication and narrative family approaches. For instance, we have seen a shift from didactic approaches where therapists were experts in charge of changing family relationships, to post-modern approaches such as Narrative Family Therapy, in which family members are considered experts on family functioning and where one goal of therapy is the recognition and use of family strengths to facilitate change.

Inevitably, theoretical models will influence to some extent where the focus of assessment lies. For example, those working within a therapy model that emphasizes the importance of cognitive processes will encourage individuals to compare their thought processes with a rational evaluation of evidence, and also to hear how other members of the family, who each undertake their own evaluations, view the matter. Thus the assessment process will focus on ascertaining family member cognitions and their evaluations of alternative explanations for events.

In contrast, where presenting difficulties pertain to family communication processes, emotional difficulties or life transition challenges, postmodern approaches will view the problem as arising out of 'oppressed' stories that dominate an individual's life and constrain the possibility of change. Assessment focuses on eliciting narratives from family members that illustrate their perspective on the matter of concern, and also the ways in which individuals see themselves in relation to each other and to the presenting difficulty. Once the dominant narrative has been identified (i.e. 'assessed'), then the way is open for a possible reauthoring (or rewriting) of the family story.

While the referral problem will influence the type of systemic approach that therapists adopt, their choice of approach will also be influenced by their personal values and the characteristics of the services in which they work. For instance, some therapists working within a legal framework may need to bear in mind that this can militate against adopting certain postmodern therapeutic practices – such as deliberately adopting a 'contrary position' – as legal frameworks have rigid rules acting as an external reality that needs to be taken into account.

Despite the various theoretical frameworks within which assessment takes place, all family system approaches share the recognition that the perceptions, emotions and behaviours of all family members are relevant. Further, that the various assessment processes will in some way seek to enable therapists and families to consider the relevance and importance of each family member's contribution to the situation with a view to identifying potential solutions to the family's concerns.

GENERAL PRINCIPLES OF FAMILY THERAPY ASSESSMENT

Several principles guide the process of assessment in modern systemic approaches. First, that an explicit theoretical framework guides the assessment process so that it will link coherently into subsequent interventions, providing 'theory–practice' links. Secondly, that the assessment process is designed to provide insights into important aspects of family functioning. Thirdly, that assessment offers opportunities for gathering baseline measures of problematic aspects of family functioning against which future outcomes can be measured [2].

GOALS OF FAMILY THERAPY ASSESSMENT

Family therapy assessment is guided by a fundamental assumption that the observation of family structure and processes as they unfold in the clinical interview will reveal something of the nature of the presenting problem, its genesis and maintenance over time. Gaining an understanding of these relationships can enable family therapists and the family to form intervention plans aimed at addressing the relevant family relationships, processes or narratives.

The question for the family therapist is how to structure the assessment in such a way that important areas of family functioning are revealed. Various assessment frameworks have been proposed [3–6]. There are differences of focus and emphasis between the various models and a variety of terms are used to capture different aspects of family functioning. Some major themes that emerge include:

- *Gaining an understanding of the presenting problem:* Families most frequently present to clinical services with a 'problem', or 'difficulty', which is regarded as 'belonging' to one family member. The family therapist will seek to gain an understanding of the complexities of the

problem – for instance, its salience for all family members, its meaning to them, their responses to it, and their views on aspects of the problems such as how and why it arose, and the effects it is having upon the family.

- *Family structure, composition and organization:* Here, the therapist will try to elicit factors such as who is in the family, the roles and responsibilities adopted by various family members, and how the family organizes and regulates itself. For instance, do the parents form a dyadic subsystem in which they are united in taking on the parenting role, or is one parent more closely aligned with other family members? Over time, how do families accommodate to internal and external forces that impact upon them? For example, all families will need to adapt to developmental child processes that lead the child from infancy to adulthood. In response to these forces for change, does the family reorganize itself in a way that is adaptive or in a way that proves maladaptive? Is the family organization stable enough for the family to respond to crises without fragmentation?
- *Family processes:* The therapist will want to elicit the nature of reciprocal interactions and transactions. That is, how family members respond to each other and how interactions at one point in time have influenced, or potentially will influence, subsequent interactions between them. Sequences of interactions will give rise to relationship patterns that the therapist will be keen to ascertain in so far as they are relevant to specific outcomes of interest.
- *Patterns of communication:* Together, family members construct an understanding of given happenings. What are these co-constructed understandings, and how do they influence the ways in which families communicate with each other? What is the affective tone of interactions – how are families conveying emotion and how are emotions influencing the communications between them?
- *Biopsychological factors:* These factors have been seen to be important in, for instance, medical family therapy models where the relationship between medical health conditions and psychosocial dimensions is explored – as, for example, in diabetes, anorexia and asthma [7]. More recently, attention has been drawn to emerging knowledge of brain chemistry, neurology and genetic factors and how these impact upon behaviour and emotions [8]. This could lead the therapist to seek to ascertain, for instance, if a child's difficulties within the school system are related to genetically influenced developmental factors. One question for the family therapist when biopsychosocial factors are of particular relevance is how do they influence both individual and family functioning, that is, what is the dynamic interrelationship between biological, psychological and family or social factors?
- *Relationship levels:* The focus here is on the social relations within the family – what are the subsystems within the larger family system, and how do these function in such a way as to account for variance between different families? These subsystems may be formalized in some models of therapy into, for instance, tracking consistent patterns of behaviour that are displayed or elicited between and from various combinations of family members, ranging from the individual level ('actor' effects), to the family as a whole (the 'group' effect) [9]. To illustrate, when examining the expression of affect within the family, how warmth is consistently expressed by a given family member constitutes an 'actor' effect, while the way in which one individual in the family elicits warmth from other family members would be regarded as a 'partner' effect [10].

Within such frameworks, information will be elicited from family members through a series of discussions between therapist and family. It is not easy, therefore, to make a sharp distinction between assessment and intervention as these discussions may, in themselves, begin to have an impact upon family communication, organization and relationship patterns.

THE PROCESS OF FAMILY THERAPY ASSESSMENT

The therapist initially meets the family and focuses upon gaining the perspectives of all family members through using both direct and circular questioning. Circular questions enable the therapist to take the feedback gained from one question and use it to shape the next and so allow for a joint construction. The style of questioning aims to draw

out similarities and differences in perspectives, and the strengths and limitations of family members; through this process the family can gain a new way of understanding and experiencing each other.

Reflecting teams are usually a part of the infrastructure of this type of therapy. They usually consist of two or three clinicians who observe the therapeutic process, with the aim of offering opinions on both the process and content of the therapy as it unfolds. They can contribute additional thoughts – for instance, with regard to observed or inferred family strengths and the meanings associated with behaviours within the family. Through this reflective process, more creative and effective intervention strategies can be generated. In addition, the reflecting team can ensure that the therapist's own responses to the family, as well as any prejudices, are managed and do not interfere with the therapeutic endeavour.

When and how the reflecting team offers their observations is dependent on the school of family therapy adopted. Some teams choose to sit behind a one-way mirror, whilst others sit in the room with the family. The team can choose to interrupt the assessment or intervention if they consider an area needs further exploration. Or they can take a prearranged break with the therapist to consider information gained so far. Feedback from the reflecting team can be fed back directly to the therapist, with the therapist feeding back in turn to the family. Alternatively, the reflecting team can discuss the assessment with each other in front of both the therapist and family, who can in this way share in their observations. These procedures provide a collaborative way for families to generate empowering solutions to their difficulties.

RESEARCH ASSESSMENT TOOLS

There are a variety of more formal direct and indirect assessment tools that can provide a picture of aspects of family functioning. These include, for instance, the Family Environment Scale [11] and the Circumplex model and Family Adaptability and Cohesion Evaluation Scale (FACES) [12]. The strengths and limitations of a wide variety of such assessment tools with respect to their utility within a child welfare context, and the ease with which they link to therapeutic models of intervention, can be found in Johnson *et al*. [13].

CONCLUSIONS

Family systems therapy offers an approach to assessment that sets an individualized presenting 'difficulty' or 'problem' firmly within its wider systemic context, offering opportunities for destigmatizing individuals, for enabling all family members to feel heard and understood, to gain new insights and recognize new meanings. It offers the potential for families to start the process of constructing new and more helpful realities. Some limitations of the approach have been noted. The majority of family therapy models use Western white culture as their main frame of reference, and the majority of therapists in the UK come from this background. While the appreciation of different cultures, religions and family forms has been acknowledged in recent years, there is a limited evidence base regarding the applicability of some systemic models to particular minority groups (e.g. single parent, gay, lesbian or blended families). An open attitude regarding the legitimacy of a range of values and attitudes across the diversity spectrum is essential if sensitive areas are to be explored in a helpful and constructive way.

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Section 7

Approaches to Intervention

41

Discovering Psychiatric Pharmacogenomics

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THE TREATMENT OF CHILDREN WITH PSYCHOTROPIC MEDICATIONS

The development of psychopharmacological interventions for the treatment of child psychiatric conditions dramatically changed the practice of child psychiatry over the course of the twentieth century. At the beginning of the century, there was not yet a single medication identified that could help children to deal with the symptoms of child psychiatric illnesses. By 1937, it was discovered that stimulants could help children with hyperkinetic behaviour [1]. Gradually, over the intervening years many other psychotropic medications have been demonstrated to have potential therapeutic benefits for children with a wide range of psychiatric disorders and symptoms.

The history of the treatment of children with severe obsessive-compulsive disorder (OCD) provides insight into the clinical appreciation of the power of new medications. Before the development of medications to treat the most extreme forms of OCD, there were many children who were treatment resistant to psychotherapy and behavioural therapy. These included quite intelligent children who had been very intensively treated. Many of the families of these children had nearly given up hope until reports of positive clinical trials in adult patients were published. Clomipramine became available in Canada before it was approved for use in the USA. During this time, US families would make regular trips to Canada, until clomipramine was eventually approved for use in the USA. The introduction of fluoxetine a few years later had another major impact on the treatment of OCD and again children treated with fluoxetine who had been intractable to any form of psychotherapeutic treatment did respond to this new medication. Of course, not all children responded to clomipramine or fluoxetine, but some children did show dramatic improvements in response to treatment with these medications. This variability in response to specific medications remains one of the greatest challenges in using psychotropic medications. The introduction of pharmacogenomic testing has provided the first important tool that is now available to improve our ability to identify a safe and effective medication for a specific patient.

NATIONAL DIFFERENCES IN THE PRESCRIPTION OF PSYCHOTROPIC MEDICATIONS FOR CHILDREN

In both the USA and Britain, the benefit of the full range of psychotropic medications has been gradually appreciated. However, it is widely recognized that the use of psychotropic medication in the USA is far more extensive than in Britain. While it is important to understand better the multiple reasons that psychopharmacological treatment is more widespread in the USA, one reason for this difference is that with more experience of prescribing psychotropic medication clinicians have personally observed the clinical benefit for some children. Given this observation, many clinicians

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have focused on this potential for positive outcome even in the light of relatively low certainty that any given child will benefit. Essentially, the potential reward of a dramatic response has been judged a sufficiently valuable benefit to justify treatment even knowing that as many as half of the treated patients will not have an effective response.

Currently, psychotropic medications play a role in the treatment of children with virtually every psychiatric diagnosis. Yet, the evidence base for the implementation of psychopharmacological treatment remains relatively subjective. Furthermore, inconsistencies in the conduct and reporting of clinical trials have led to considerable controversy. At the heart of this dialogue is the clinical observation that many children respond dramatically to psychotropic medications while other children do not respond at all or, in fact, get worse. Despite years of investigation, it has only been in the last decade that our understanding of some of the biological factors responsible for these variable responses has begun to come into focus.

There are two major issues that have slowed the adoption of psychopharmacological treatment in both the USA and Britain. The first is the recognition that there are a wide range of adverse drug effects that occur in patients treated with any class of psychotropic medications. The second is an almost universal appreciation that there is wide individual variability in the response of children to treatment with psychotropic medications. After years of using empirical approaches to guide the use of psychotropic medications for child psychiatric illnesses, there is now an evidence-based methodology to aid clinicians both to select medications and to predict the appropriate dosage for a specific patient.

WHAT IS PSYCHIATRIC PHARMACOGENOMICS?

Psychiatric pharmacogenomics is a scientific approach to using the measurement of genetic variability to predict the medication response of a specific child. Pharmacogenomics is not an easy discipline to approach for psychiatrists who are not familiar with molecular genetics, but it can be mastered by systematically working through the basics of how changes in gene structure influence gene function [2].

Schoolchildren in the USA and Britain learn that both the Rosetta stone and the genetic code are important examples of famous breakthroughs in our understanding of the world. The Rosetta stone represents an ability to use a familiar language to translate a cryptic form of writing of the past. Breaking the genetic code allowed us to translate the language of nucleotides in a way that provides an understanding of the nature of individual genetic variability. What has been amazingly slow to be realized is that variations in the genetic sequences of our patients give us the ability to predict their responses to psychotropic medications.

As outlined in the previous section, we have successfully developed medications to treat the symptoms of child psychiatric disorders. In addition to the selective serotonin reuptake inhibitors (SSRIs) that are used for mood disorder and OCD, we also have stimulants and atomoxetine for attention deficit disorder. The atypical antipsychotics are used for schizophrenia and bipolar disorder, which is also treated with lithium and other mood-regulating medications. While clinicians have learned that these medications can be extremely effective for some children, the sobering reality is that they can also make other children much worse. Until very recently, there was no other alternative but to proceed with a 'trial-and-error' strategy to search for the right medication for a particular child.

An appreciation of how predictions of medication response can be derived requires learning about gene structure and function. The most straightforward examples are genotyping drug metabolizing genes, such as the cytochrome P450 2D6 gene, that produce enzymes that metabolize psychotropic medications (Table 41.1).

Table 41.1 The 2D6 relative role in the metabolism of common antidepressants.

Primarily metabolized by 2D6	Substantially metabolized by 2D6	Minimally metabolized by 2D6
Desipramine	Amitriptyline	Citalopram
Doxepine	Bupropion	Desvenlafaxine
Fluoxetine	Duloxetine	Escitalopram
Nortriptyline	Imipramine	Fluvoxamine
Paroxetine	Mirtazapine	Sertraline
Venlafaxine	Trazodone	

THE CYTOCHROME P450 2D6 GENE

The cytochrome P 450 2D6 gene (*CYP2D6*) has been studied for the last 30 years and is only one of dozens of genes that can provide insight into the response of a patient to a medication [3]. However, the beginning of child psychiatric pharmacogenomics emerged from the study of variations in drug metabolism of 2D6 substrate medications. For child psychiatrists, the testing of *CYP2D6* allows them to determine with a high degree of certainty whether a particular child will be able to tolerate a medication like paroxetine, fluoxetine, atomoxetine, risperidone or haloperidol as a consequence of their ability to metabolize these medications.

The *CYP2D6* gene is located on the smallest autosome, which is chromosome 22. Its chromosomal address is 22q, which indicates that it is on the long arm of this short chromosome. It is composed of 1491 nucleotides and has nine exons that code for an enzyme with 497 amino acids. It is one of the most highly variable genes that are commonly genotyped. The more than 100 variants are catalogued on the Karolinska Institute website (http://www.cypalleles.ki.se/). Essentially, it is possible to identify children who are either slow metabolizers or fast metabolizers.

Poor 2D6 substrate metabolizers

Children who lack even one 'good' copy of the *CYP2D6* gene are classified as poor metabolizers. These children cannot make enough 2D6 enzyme to metabolize standard doses of fluoxetine or paroxetine. Approximately 10% of patients of European ancestry are poor metabolizers and experience moderate to severe side effects at standard doses of these drugs. Both children and adults who are poor metabolizers of 2D6 have had fatal toxic reactions to 2D6 substrate medications [4,5].

The case of a 9-year-old boy who was treated with up to 100 mg of fluoxetine for the treatment of OCD with comorbid Tourette's disorder has been described [4]. The boy developed status epilepticus and then died of a cardiac arrest. It was subsequently determined that he had two inactive copies of the *CYP2D6* gene. At autopsy, it was determined that his serum fluoxetine and serum norfluoxetine levels were both in the toxic range. This provided evidence that the elevated serum fluoxetine level was not the result of an acute overdose of fluoxetine. Pharmacogenomic testing would have revealed that this child was at high risk for a fatal outcome if given high doses of fluoxetine.

Ultra-rapid 2D6 substrate metabolizers

Children who have three or more active copies of the 2D6 gene or who have two or more copies of the upregulated alleles of *CYP2D6* have been demonstrated to metabolize 2D6 substrates very quickly. These children are usually unable to achieve therapeutic serum levels of 2D6 substrate medications at traditional doses. The demonstration of this very rapid metabolism has been documented by pharmacokinetic studies that have revealed significantly decreased drug exposure in patients with ultra-rapid metabolism [6].

Child psychiatrists must also become aware of the implications of the *CYP2D6* metabolic capacity of the mothers of breastfed infants, as the mental status of these infants can be dramatically affected if these mothers take prodrugs like codeine. A breastfeeding mother who is an ultra-rapid metabolizer of 2D6 substrate medications will rapidly metabolize codeine to morphine. Given that high serum levels of morphine in the mother will result in high levels of morphine in her breast milk, her infant will become increasingly lethargic as a consequence of nursing. A tragic case of an infant who died as a consequence of morphine toxicity was reported in the *Lancet* and has fortunately led to much greater awareness of the value of pharmacogenomic testing in pregnant women [7].

BEYOND CYP2D6

Given that there are many gene variations that affect both drug metabolism and response, psychiatric pharmacogenomic testing is increasingly assessing variation in many relevant genes that ultimately influence how a particular patient will respond to a specific medication. It is increasingly apparent that clinicians will need to systematically review the implications of these gene variations for the medications that they prescribe [2].

Currently, it is possible to order the genotyping of both drug-metabolizing enzyme genes and key target genes such as the serotonin transporter (*SLC6A4*) and the serotonin receptor genes (*HTR2A, HTR2C*) to guide the selection and dosing of psychotropic medications. Other

informative genes include the catecholamine-*O*-methyltransferase gene (*COMT*) and the dopamine receptor genes (*DRD2, DRD3, DRD4*).

It is clearly most practical for clinicians to order these pharmacogenomic panels from laboratories that provide comprehensive guidance on the implications of these gene variations for specific psychotropic medications. Fortunately, there are now multiple reference laboratories that are providing pharmacogenomic testing and interpretations.

FUTURE EXPECTATIONS FOR PHARMACOGENOMICS TESTING

Many child psychiatrists in the USA have begun to adopt clinical pharmacogenomic testing [8]. However, pharmacogenomic testing is still largely undiscovered in Britain. This is almost certainly the direct result of the much wider use of psychotropic medications in the USA. However, it is safe to predict that within the next decade there will be rapid adoption of psychiatric pharmacogenomic testing on both sides of the Atlantic. The most important reason is that gene sequencing will become available as a standard component of a comprehensive clinical diagnostic evaluation. Today, in order for child psychiatrists to order a panel of informative pharmacogenomic genes, they must have a cognitive understanding of the rationale for the potential benefit of testing. Of course, there is also a consideration about whether the benefit is worth the cost of the testing. When the cost of genotyping the entire genomic sequence of a patient falls below US$1000, sequencing will become a standard component of a comprehensive patient evaluation. The current director of the National Institute of Health in the USA has predicted that patients will be routinely sequenced when the cost of sequencing reaches $1000. Furthermore, he estimates that this price point will be reached by 2015 [9]. After 2015, it will still be necessary to develop software to interpret the implications of the genetic variability of our patients, but there will be a stampede of bioinformaticists who will be competing to develop the most effective methodologies to diagnose and treat both the traditional physical illnesses as well as what we currently refer to as mental illnesses.

It is likely that in the next five years the genotyping of panels of pharmacogenomically informative genes will become increasingly commonplace in the USA. As the number of adverse effects of psychotropic medications decreases, the use of these medications will, in all likelihood, increase even in children with less severe psychiatric illnesses. Ultimately, the societal cost of mental illness will drop as the chronic disabilities that we currently manage become more of a historical memory than our daily responsibility.

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INTERNET RESOURCE

Karolinska Institute, Human Cytochrome P450 (CYP) Allele Nomenclature Committee: http://www.cypalleles.ki.se/.

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Cognitive–Behavioural Therapy for Children and Adolescents

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Cognitive–behavioural therapy (CBT) is a treatment approach based on the general notion that a psychological disorder is caused or maintained by 'dysfunctional' thought patterns and lack of positively reinforced adaptive behavioural coping strategies. CBT is a class of treatment; all cognitive–behavioural treatments aim to identify and reduce cognitive biases or distortions and build effective coping and problem-solving skills. After decades of extensive research on CBT in adult populations [1], CBT is now being applied to child and adolescent populations with success.

BASIC PREMISES OF THE CBT APPROACH AND ITS ADMINISTRATION

According to the basic CBT model, disorder is conceptualized as resulting, in part, from the individual's cognitive distortions (such as false attributions or expectations of the self or other) that undermine positive coping and problem-solving behaviour. There is now considerable evidence that cognitive distortions exist and may play a causal – or at least contributing – role in many childhood disorders, with much of the work focusing on depressed, anxious and conduct-problem youth [2–5] (Box 42.1). Several clinical and developmental models have informed and been informed by research into the processes by which distorted cognitions are developed and influence behavioural/emotional problems. One example, the social information processing model [5], was developed in the context of conduct disorder but has proved useful for other childhood disorders, including depression and anxiety. The model focuses on the following:

- the child's attending to, encoding and interpreting social cues (e.g. why did that child step on my foot?);
- developing goals for one's own behaviour (e.g. what do I want to do now?);
- generating potential solutions and evaluating their effects (e.g. what would happen if I hit back at him?).

Several CBT programmes have been devised and shown to be clinically effective. Although there is some tendency to tailor the CBT treatment for a particular disorder (see studies cited below), it is possible to make several basic statements about how CBT is administered. In general, CBT interventions seek to break the cascade of maladaptive thoughts and feelings that lie between the cognitive distortion and the destructive behaviour. This occurs in a logical, stepped manner usually lasting 8–16 sessions, typically on a 1 session/week schedule (Box 42.2). A first step is to collate detailed information about the settings that lead the child to feel, for example, anxious and unable to cope with a particular situation. A second step is to help the child/adolescent to identify and differentiate thoughts, feelings and somatic reactions linked with these situations Subsequently, there is a focus on self-talk, or helping the child to recognize

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Box 42.1 Examples of key cognitions associated with childhood

Characteristics of clinically anxious children [3]
- Vigilant to threat
- Interpret ambiguity as more threatening
- Come to faster conclusions about threat
- Underestimate personal coping ability
- Anticipate distress (often exaggerated) in the face of threat

Characteristics of clinically aggressive children [4]
- Attend to less social cues
- Direct attention towards hostile social cues
- Interpret stimuli in a hostile manner
- Generate fewer solutions to social problems
- Positively appraise aggressive responses
- Positively appraise own ability to perform aggressive response

Characteristics of clinically depressive children [5]
- Selectively attend to negative features of events
- Report negative attributions (i.e. internal, stable explanations for positive events and external, unstable explanations for negative events)

Box 42.2 Core steps in cognitive—behavioural therapy (CBT) for childhood anxiety

- Recognize feelings and physical reactions
- Identify associated thoughts (e.g. interpretations, attributions and expectations)
- Cognitive restructuring/coping self-talk
- Progressive muscle relaxation
- Imaginary/*in vivo* (graded) exposure
- Self-evaluation and reward

how certain kinds of self-talk can be destructive ('I'll look silly') and promoting positive self-talk ('I have done this OK before'). Using these skills, children are then supported to develop a hierarchy of anxiety-producing situations, which they gradually face, with a clear reward structure in place. Relaxation is often included to improve the child's coping strategies and expand his/her coping repertoire. Throughout treatment children are helped to evaluate their newly developed coping skills in 'real-life' settings, and these are rewarded where appropriate. This can continue for several sessions, as the child learns to test new strategies and, through trial and error, to find strategies that work and to diagnose why other strategies do not. Homework throughout the treatment process fosters understanding of why feelings of anxiety or depression develop and how they might be managed effectively. In addition, emphasis is placed on developing rapport with the child/adolescent throughout the treatment. CBT programmes value rapport, but unlike some approaches, do not construct the treatment as working through the relationship with the therapist. Instead, the CBT therapist guides the child/adolescent to reshape attributions and expectations in order to change behaviour. Treatments including a family component are increasingly common, and typically this means an ancillary focus on the parents' behaviour, or a parent's own anxiety

and how that may influence the child via the child–parent relationship. These approaches build on and address the finding that parental anxiety has been found to be a significant predictor of treatment failure of individual treatment of the child [6]; parents who model poor coping, parent in an overprotective manner and communicate expectations to the child that she/he cannot cope with effectively may undermine the child's individual treatment [7]. Particularly valuable in this area are studies now underway that seek to manipulate the degree of family involvement in order to better understand treatment mechanisms. It is not yet clear what additional benefits are conferred by these treatment models, but findings from these studies should be closely monitored because they may have a substantial impact on how CBT-related treatments for children may be optimized.

DEVELOPMENTAL CONSIDERATIONS

We now know that depressed, anxious or conduct-problem-related cognitions are evident at an early age. In a study of 5-year-olds, Murray and colleagues found that higher rates of negative cognitions, defined as 'spontaneous' expressions of hopelessness or low self-worth during an experimentally manipulated card game with a friend, were observed in children whose mothers were, or had been, depressed [8]. Significantly, differences between the children of depressed and non-depressed mothers were apparent only when the children were losing. Evidence that cognitive distortions do not operate in a trait-like manner, even in 5-year-olds, is an important clinical and developmental lesson. Other studies also suggest that cognitive biases or distorted 'filters' exist in young children and may be learned from parent-child interactions. A study of 2–6$\frac{1}{2}$-year-olds found that insecurely attached children showed poorer understanding of negative emotions compared with securely attached children; in other words, they had more difficulty explaining or making sense of negative emotions [9]. Findings from these and many other studies are valuable not only for what they say about the phenomena, but also for the practical lessons they yield for assessing young children. Greater integration of these methods in clinical settings is feasible and a valuable next step for advancing clinical assessment and treatment monitoring (Box 42.3). Demonstrating that young children with elevated behavioural/emotional symptoms exhibit cognitive distortions does not mean that these cognitive processes are causally linked with disorder; neither does it necessarily imply that altering these cognitions will produce positive behavioural change. Indeed, it is somewhat surprising that little is known about the developmental constraints around CBT-based treatments, and clinical research has not yet demonstrated that a child's developmental stage predicts treatment outcome. This may be because the predictors so far considered (e.g. age) are weak indicators of the cognitive and social processes that are required for successful CBT. However, the theory and implementation of CBT has not been especially developmentally informed. So, for example, the traditional CBT model is not explicit about why the approach might work with a 12-year-old but not with a 5-year-old. If there is a general impression, it is that CBT is an effective treatment for depression and anxiety in children aged around 8 years, with both short- and long-term gains. In any event, it is clear that CBT may be very effective. It is worth noting that Kendall and Southam-Gerow found that individual CBT was highly effective in treating children/adolescents with anxiety disorders, and that approximately 90% were diagnosis-free more than 3 years after treatment ended [10].

RECENT ADVANCES IN CBT PROGRAMMES FOR CHILDREN AND ADOLESCENTS

Recent clinical research findings on CBT in children and adolescents are noteworthy in several respects. One is the enlarged range of conditions for which CBT has produced large, reliable and clinically meaningful findings. So, for example, in addition to depression and anxiety [10,11], there are now studies showing positive effects for post-traumatic stress disorder [12,13] and obsessive-compulsive disorder [14]. However, it remains the case that most of the CBT studies are efficacy studies, that is, the treatment has been shown to work under relatively controlled conditions. As a result, concerns about the generalizability of the study effects have been expressed. These are real and important, but they should not be seen as reasons for not undertaking standard treatment protocols. In any event, what is needed

Box 42.3 Sample responses to ambiguous scenarios

You have arranged to have a party at 4 pm and by 4.30 no one has arrived

Cognitive bias — What do you think is most likely to have happened?

- Anxious: 'Nobody wants to come to the party'
- Aggressive: 'Nobody wants to come to the party'
- Non-clinical controls: 'They might be late because there is bad traffic'

Behaviour — What will you do about it?

- Anxious: 'Nothing. Feel upset'
- Aggressive: 'Get cross and when I see them at school I will tell them I don't want to be friends with them'
- Non-clinical controls: 'Phone around and see where they are and when they will arrive'

You are playing inside and your dog starts barking and growling outside

Cognitive bias — What do you think is most likely to have happened?

- Anxious: 'There is someone I don't know trying to get into my house'
- Aggressive: 'Someone is stealing my bike from outside'
- Non-clinical controls: 'Another dog is walking past outside'

Behaviour — What will you do about it?

- Anxious: 'Hide'
- Aggressive: 'Find the thief and hit them'
- Non-clinical controls: 'Look out of the window and tell my dog to be quiet'

Adapted from Barrett PM, Rapee PM, Dadds MR, Ryan SM. Family enhancement of cognitive style in anxious and aggressive children. *Journal of Abnormal Child Psychology* 1996;24:187–203.

now are studies that carry out CBT-based interventions for child populations in conventional clinical settings using samples that are representative of clinic settings. Work of that kind is underway. In a recently completed study of 41 children with anxiety disorder treated within a primary care setting, we found equivalent outcomes (61% free of primary anxiety diagnosis post-treatment) to those found in trials conducted in specialist child anxiety clinics [15]. These preliminary findings are promising and encourage the application of these protocols in non-specialist settings.

Another recent advance in work on CBT in children and adolescents is that it is increasingly being set up against or in addition to medication. In fact, there are several large-scale trials comparing CBT with drug and combined conditions. Probably the best known are the Treatment for Adolescents with Depression Study (TADS [11]) and the Child Anxiety Multisite (CAM [16]) study. In the TADS study, follow-up to 36 weeks shows that treatment-group differences apparent in the earlier phases of treatment diminished over time, with the result that there was convergence among the CBT-only, medication-only (fluoxetine), and CBT plus medication conditions [11]. The rate of adolescents with suicidal ideation (none committed suicide in the trial) was considerably higher in the medication-only group (15%) than in the combined (8%) or CBT only (6%) conditions; that is naturally a major consideration when making treatment decisions. Perhaps even more impressive are data showing that CBT can be effective as a treatment strategy even where drug treatment was ineffective [17]. However, this study did not include a CBT-only arm, and so it is not

possible to know whether CBT alone would have been successful following drug treatment failure. The CAM study, in contrast, compared outcomes of children aged 7 to 14 years with a diagnosed anxiety disorder randomized to either 14 sessions of CBT, sertraline, a combination of sertraline and CBT, or a placebo drug [16]. Based on a clinician's global impressions of improvement, 81% of children were reported as 'much' or 'very much' improved following the combination treatment, 60% for CBT, and 55% for sertraline, all of which were superior to outcomes from placebo (24%). Importantly there was not a greater frequency of adverse events amongst the sertraline group; however, there were significantly greater incidence of symptoms of insomnia, fatigue, sedation and restlessness amongst children who received sertraline in comparison to CBT.

These findings and those of other studies have shown that assumptions about CBT being appropriate only for more mild cases is no longer supported by the evidence. Indeed, although its impact is even greater when coupled with medication, CBT is an important stand-alone treatment and, in terms of its effectiveness and side-effect profile, it can be considered as a first-choice treatment rather than an add-on to other approaches, such as medication. The question now for mental health professionals is not whether CBT is a reasonable treatment option, but rather how to increase the availability of CBT to children and families. Another recent advance in CBT work concerns the mediators of treatment (why does treatment work?) and the moderators of treatment (what predicts who will respond and who will not?). Findings from this research are important for improving our understanding of the treatment process and for better targeting those who are most likely to benefit from treatment. There are suggestions but, to date, not much consistency across studies. This is likely to improve as research studies move on from asking if treatment works and take on the next stage of research, namely questions about why and for whom treatment works.

Finally, CBT is incorporated in prevention programmes in universal, indicated and selected samples. A recent review of school-based programmes [18] indicated that they were effective for anxiety, with effect sizes ranging from small to large. That is significant because it demonstrates that CBT can be used as a general tool and across a variety of settings.

CONCLUSION

Children's cognitions about their social world reflect developmental histories that shape behaviour. CBT is concerned with how these cognitive processes may be altered and, when altered, if there are consequential reductions in psychiatric symptoms and improvements in social functioning. Results from many clinical trials have shown that CBT reliably improves outcomes in children; recent studies show that CBT should no longer be considered only for more mild cases, and that the list of targeted disorders appropriate for CBT is increasing. Additionally, CBT has been shown to be as effective as, perhaps better than, and possibly a useful adjunct to, medication. These findings are important for instilling confidence in recommending treatment for ill and distressed children and their families. A next step is to develop increasingly efficient modes of delivery to improve access to this effective form of treatment.

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43

Parenting Programmes for Conduct Problems

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EVIDENCE LINKING PARENTING TO CHILD PSYCHOPATHOLOGY

The finding that parent–child relationship quality is associated with aggressive behaviour, conduct disorder and delinquency is one of the most widely reported in the literature, repeatedly found in large-scale epidemiological investigations, intensive clinical investigations and naturalistic studies of diverse samples using a mixture of methods [1]. The sort of parenting behaviours associated with these outcomes are high criticism and hostility, harsh punishment, inconsistent discipline, low warmth, low involvement, low encouragement and poor supervision.

The link with depression, anxiety and other emotional problems (e.g. somatic complaints, social withdrawal) is clear, although smaller than that found for disruptive outcomes [2]. There is also a connection between parenting and quality of a child's peer relationships, mediated by social cognitions and behavioural strategies learned from interacting with parents (Box 43.1).

PROGRAMMES FOR CHILDREN BASED ON SOCIAL LEARNING THEORY

Programmes based on social learning theory have evolved for more than 40 years and there is a large evidence base. Most are aimed at antisocial behaviour as their proximal target outcome. The content and delivery of a typical programme is shown in Box 43.1. Most basic programmes take 8–12 sessions, lasting 1.5–2 hours each. Full accounts of programmes are given by the developers [3,4].

FORMAT OF A TYPICAL SOCIAL LEARNING PROGRAMME

Teaching a child-centred approach

The first session covers play. Parents are asked to follow the child's lead rather than impose their own ideas. Instead of giving directions, teaching and asking questions during play, parents are instructed simply to give a running commentary on their child's actions. As soon as the parent complies, the practitioner gives feedback. After 10–15 minutes, this directly supervised play ends and the parent is 'debriefed' for half an hour or more alone with the clinician.

The second session involves elaboration of play skills. The previous week's 'homework' of playing at home is discussed with the parent in considerable detail. Often there are practical reasons for not doing it ('I have to look after the other children, I've got no help') and parents are then encouraged to solve the problem and find ways around the difficulty. For some parents there may be emotional blocks ('it feels wrong – no one ever played with me as a child'), which need to be overcome before they feel able to practise the homework.

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Box 43.1 Features of effective parenting programmes based on social learning theory

Content

- Structured sequence of topics, introduced in set order during 10–12 weeks
- Curriculum includes play, praise, rewards, setting limits and discipline
- Parenting seen as a set of skills to be deployed in the relationship
- Emphasis on promoting sociable, self-reliant child behaviour and calm parenting
- Constant reference to parent's own experience and predicament
- Theoretical basis informed by extensive empirical research and made explicit
- Plentiful practice, either live or role-played during sessions
- Homework set to promote generalization
- Accurate but encouraging feedback given to parent at each stage
- Self-reliance prompted (e.g. through giving parents tip sheets or book)
- Emphasis on parents' own thoughts and feelings varies from little to considerable
- Detailed manual available to enable replicability

Delivery

- Strong efforts made to engage parents (e.g. home visits if necessary)
- Collaborative approach, typically acknowledging parents' feelings and beliefs
- Difficulties normalized, humour and fun encouraged
- Parents supported to practise new approaches during session and through homework
- Parent and child can be seen together, or parents only seen in some group programmes
- Creche, good-quality refreshments, and transport provided if necessary
- Therapists supervised regularly to ensure adherence and to develop skills

After this discussion, live practice with the child is carried out. This time the parent is encouraged to go beyond describing the child's behaviour and to make comments describing the child's likely mood state (e.g. 'you're really trying hard making that tower', or 'that puzzle is making you really fed up'). This process has benefits for both the parent and the child. The parent gets better at observing the fine details of the child's behaviour, which makes them more sensitive to the child's mood. The child gradually gets better at understanding and labelling his/her own emotional states.

Increasing desirable child behaviour

Praise and rewards are covered here. The parent is required to praise their child for lots of simple everyday behaviours such as playing quietly on their own, eating nicely, and so on. In this way the frequency of desired behaviour increases. However, many parents find this difficult. Usually, with directly coached practice, praise becomes easier. Later sessions go through the use of reward charts.

Imposing clear commands

A hallmark of ineffective parenting is a continuing stream of ineffectual, nagging demands for the child to do something. Parents need to be taught to reduce the number of demands, but make them much more authoritative. This is done through altering both the manner in which they are given, and what is said. The manner should be forceful (standing over the child, fixing him/her in the eye, and in a clear firm voice giving the instruction).

The emotional tone should be calm, without shouting and criticism. The content should be phrased directly ('I want you to . . . '). It should be specific ('keep the sand in the box') rather than vague ('do be tidy'). It should be simple (one action at a time, not a chain of orders), and performable immediately. Commands should be phrased as what the parent does want the child to do, not as what the child should stop doing ('please speak quietly' rather than 'stop shouting'). Instead of threatening the child with vague, dire consequences ('you're going to be sorry you did that'), 'when–then' commands should be given ('when you've laid the table, then you can watch television').

Reducing undesirable child behaviour

Consequences for disobedience should be applied as soon as possible. They must always be followed through: children quickly learn to calculate the probability that consequences will be applied, and if a sanction is given only every third occasion, a child is being taught he/she can misbehave the rest of the time. Simple logical consequences should be devised and enforced for everyday situations (e.g. if a child refuses to eat dinner, there will be no pudding). The consequences should 'fit the crime', should not be punitive, and should not be long term (e.g. no bike riding for a month), as this will lead to a sense of hopelessness in the child, who may see no point in behaving well if it seems there is nothing to gain. Consistency of enforcement is central.

Time-out from positive reinforcement remains the final 'big one' as a sanction for unacceptable behaviour. The point here is to put the child in a place away from a reasonably pleasant context. Parents must resist responding to taunts and cries from the child during time-out, as this will reinforce the child by giving attention. Time-out provides a break for the adult to calm down also.

INTERVENTIONS WITH YOUTH

In adolescence somewhat different approaches are necessary, with more emphasis on negotiation and close supervision when the young person is out of the home. Also, whilst many components of programmes based on social learning theory are incorporated, additional elements may be required. In particular, there may need to be more of a focus on the wider systems around the youth, be they the wider family, school or peer networks. Thus interventions tend to be one of two types: family-based interventions or multicomponent interventions.

Family-based interventions

Being based on systemic family therapy theories, family-based interventions typically attempt to alter the structure and functioning of the family unit. The best known in the context of delinquency is Functional Family Therapy (FFT) [5]. It is designed to be practicable and relatively inexpensive: 8–12 one-hour sessions are given in the family home, to overcome attendance problems common in this client group; for more intractable cases, 26–30 hours are offered, usually over 3 months.

There are three phases to treatment; the first is the *engagement and motivation* phase. Here the therapist works hard to enhance the perception that change is possible. The aim is to keep the family in treatment, and then to move on to find what precisely the family wants. Techniques include reframing, whereby positive attributes are enhanced (e.g. a mother who continually nags may be labelled as caring, upset and hurt). The next phase is not commenced until motivation is enhanced, negativity decreased, and a positive alliance established.

The second phase targets *behaviour change*. There are two main elements to this: communication training and parent training. This stage is applied flexibly according to family needs. Thus if there are two parents who continually argue and this is impinging on the adolescent, the 'marital subsystem' will be addressed, using standard techniques. Parent training techniques are similar to those found in standard approaches.

The third and final phase is *generalization*. Here the goal is to get the improvements made in a few specific situations to generalize to other similar family situations and to the wider community. For example, to help the youth and family negotiate positively with community agencies such as school and to help them get the resources they need. Sometimes this latter goal may require the therapist to be a case manager for the family.

Multicomponent interventions

These attempt to target multiple risk factors in multiple domains, with the best known being Multisytemic Therapy (MST) [6]. The initial focus of

MST is an assessment that will identify the youth's difficulties in relation to the wider environment. Difficulties are understood as a reaction to a specific context, not seen as necessarily intrinsic deficits. At the same time strengths will be identified that can be used as levers for positive change. These may be in the young person, the parents, the wider family, peers, the school or the community.

Interventions are designed to promote responsible behaviour and decrease irresponsible behaviour with the aim of helping the youth become independent and develop prosocial life skills. They will be focused in the present and be action oriented with well-defined specific goals. This requires daily or weekly efforts by family members, which enables frequent practice of new skills, positive feedback for efforts made and rapid identification of non-adherence to treatment.

Intervention effectiveness is evaluated continually with the intervention team assuming responsibility for overcoming barriers to successful outcomes. Whilst the way the therapy is delivered is closely controlled, the precise nature of moment-to-moment interaction is not tightly prescribed. In a sense MST is a set of operating principles that draw on the evidence for whatever works – e.g. cognitive–behavioural therapy (CBT), close monitoring and supervision – rather than one specific therapy.

EFFECTIVENESS

Social learning approaches

Systematic reviews and meta-analyses of studies usually with 'no treatment controls' confirm that these approaches work well for antisocial children aged 3–10 years [7]. Mean effect sizes across studies vary from around 0.4 to 1.0 according to outcome, thus showing good effectiveness.

Youth interventions

Adolescents are generally found to do less well in parenting programmes for antisocial behaviour. However, studies on adolescents generally have the most severe, persistent cases. When cases of similar severity are compared directly there is no age effect [8]. The results for FFT and MST are reasonably impressive, at least in the USA [9,10]. Whilst evaluations outside the USA are either lacking or mixed there are currently UK trials of these interventions underway.

MEDIATORS OF CHANGE

In recent years, researchers have begun to investigate the factors that mediate outcome. This research helps to identify the 'active ingredient' of therapy. Both reductions in negative parenting (critical, harsh and ineffective practices) [11] and increases in positive parenting [12] have been shown to mediate a reduction in child symptoms.

DISSEMINATION: THE ROLE OF THERAPIST SKILL

Therapist performance can be divided into three parts: the alliance, which could be defined as how well, both personally and collaboratively, the client and therapist get on together; fidelity or adherence to specific components of a model, which concerns the extent to which the therapist follows the actions prescribed in the manual; and the skill or competence with which the therapist carries out the tasks (i.e. how well the therapist performs the actions). A meta-analysis of youth studies found that the alliance contributed on average an effect size of 0.21 standard deviations to outcome; this finding held across treatment types, and across youth, parent and family approaches [13]. In a trial under regular clinical conditions [14], therapist skill had a large effect on child outcomes – the worst therapist made outcomes slightly worse. These findings have major implications for service delivery, since they suggest that at least for multi-problem, clinical cases a high level of therapist skill is required, and staff training will need to reflect this.

CONCLUSION

The best parenting programmes incorporate empirical findings from developmental studies and are effective in using these to alter dimensions of parenting, which in turn improve child outcomes. In future, better assessments of parenting are needed so that programmes can be tailored to specific needs rather than 'one size fits all'.

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44

Systemic and Family Approaches to Intervention

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INTRODUCTION

The word 'systemic' holds many meanings that have changed and evolved over time according to historical and political contexts [1]. With practice ever-changing and evolving, approaches that have been researched are unlikely to be at the cutting edge of practice. As Carr [2] notes, most of the approaches researched thus far belong to modernist early systemic approaches rather than to later postmodern approaches that are less amenable to manualizing and randomized controlled trials (RCTs). Modernism here refers to the rationalist, materialist and reductionist view that an objective understanding of a shared, universal and measurable reality is achievable. In contrast postmodernist approaches see our understanding of the world as tentative and provisional, elaborated by individuals within particular communities. Such approaches are inherently sceptical about the universal applicability of any treatment approach.

Research trials assessing the effectiveness of family-based approaches to common problems exhibited by children and young people have recently been reviewed [2]. Here I focus on the most promising approaches, before noting evidence of treatment effectiveness of more recent postmodernist practices drawn from a different research tradition.

EXTERNALIZING DISORDERS

Attention deficit hyperactivity disorder (ADHD)

Systemic interventions for ADHD comprising sessions with families, school staff and young people, are best offered as elements of multimodal programmes involving stimulant medication [3], with systemic interventions playing an increasingly important role in the longer term [4]. Family therapy for ADHD focuses on helping families to develop patterns of organization conducive to effective child management: a high level of parental cooperation; clear intergenerational boundaries; warm, supportive family relationships; clear communication and clear, moderately flexible rules, roles and routines [5].

Conduct problems in adolescence

Woolfenden *et al.* [6] found that family-based interventions were more effective than routine treatment – falling on a continuum of care extending from Functional Family Therapy through more intensive Multi-Systemic Therapy (MST), to very intensive treatment foster care. The first two of these approaches are discussed elsewhere in this volume (see Chapter 43), so I will only discuss here the third and most intensive of these interventions, Multi-Dimensional Treatment Foster Care.

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Multi-Dimensional Treatment Foster Care (MDTFC): This approach aims to help adolescents with pervasive conduct problems and their families by linking both to a new and positive familial system: a treatment foster family. It aims to modify problem-maintaining factors in all systems by placing the adolescent temporarily within a foster family in which the foster parents have been trained to use behavioural strategies to modify the youngster's deviant behaviour. The goal is to avoid long-term separation so that as therapeutic progress is made, adolescents spend more time with their natural family. Chamberlain and Smith [7] in a review of two studies found that compared with care in a group home for delinquents, this treatment approach reduced running away from placement, rearrest rate and self-reported violent behaviour. Benefits were due to improvements in parents' skills for managing adolescents in a consistent, fair and non-violent way, and to reductions in adolescents' involvement with 'deviant' peers, with cost savings of $40,000 per case in juvenile justice and crime victim costs.

It is clear that the family-based interventions described in Wolfenden *et al*. [6] can be effective with adolescents in contact with juvenile justice systems. The authors note, however, a lack of RCTs for family and parenting interventions for children and adolescents with conduct disorders, who have had no contact with juvenile justice systems [6]. In addition they point out that in the studies entering into their review, there was insufficient evidence of beneficial effects on problem behaviour, parental mental health, family functioning and peer relations. Long-term follow-up on adult outcomes was not available.

Transferability of treatment approaches: The transferability to a UK setting of these multidimensional systemic treatment approaches developed in the USA has yet to be established. In a London borough trial of MDTFC the project team had great difficulty with respect to two treatment protocol conditions: recruiting two-parent foster families and persuading adolescents to give up their mobile phones. Holmes *et al*. [8], however, have reported some early positive findings from this British trial: the social care costs incurred by the sample children in the first 6 months of the pilot study were about 15% less than those they had incurred in the 6 months prior to entry.

All of these treatment approaches are labour intensive: treatment foster care is offered for a period of up to a year, followed by an ongoing multisystemic intervention. During a time of reductions in public services in the UK, evidence drawn from current RCTs will need to provide compelling evidence of effectiveness to justify such costly investment.

Substance misuse in adolescence

Liddle [9] found that family therapy with young people who misuse substances was more effective than routine individual or group psychotherapies in engaging and retaining them in therapy, and in improving psychological, educational and family adjustment. Liddle's version of multidimensional family therapy involves assessment and intervention in four areas:

- the adolescent as an individual *and* a member of a family and peer network;
- the parent(s) – both as individual adults and their roles as mother, father or caregiver;
- the family environment and family relationships, as manifested in day-to-day family transactional patterns;
- extrafamilial sources of influence such as peers, school and juvenile justice.

Interventions are made within and coordinated across domains, with progress in one area or with one person having implications for others. Individual meetings with parent(s) and teenager set the stage for family sessions, and family meetings may offer content and new outcomes to take to meetings with juvenile justice or school personnel. Liddle emphasizes that this approach was developed and tested as a *treatment system* rather than a one-size-fits-all approach [10]. That is, as a system offering different versions of a clinical model that vary according to factors such as clinical sample characteristics (e.g. older versus younger adolescents), and treatment parameters (e.g. type of clinical setting).

EMOTIONAL PROBLEMS

Anxiety

Systematic reviews (e.g. Ref. [11]) show that a family-based treatment for anxiety disorders is at least as effective as individual cognitive–behavioural therapy (CBT) in alleviating

symptoms of anxiety, and more effective where parents also have anxiety disorders, and in improving the quality of family functioning.

Encopresis

In a narrative review of 42 studies McGrath *et al*. [12] found that multimodal programmes involving medical assessments and intervention followed by behavioural family therapy were effective for 43–75% of cases. Effective behavioural family therapy involves psycho-education coupled with a reward programme. There is some evidence [13] that a narrative approach is more effective than a behavioural one. In the former, child symptoms are 'externalized', that is, they are talked about in such a way that they are no longer seen as 'belonging' to the child. This can help children feel less blamed and stigmatized and more in control. In a retrospective study of 108 children with soiling problems the 54 who were treated using an externalizing approach did better than children receiving a standard behavioural intervention, with parents rating the externalizing intervention as much more helpful.

Depression

Effective family-based interventions for children and adolescents with depression aim to decrease family stress and enhance social support within the family context through the facilitation of clear parent–child communication, the promotion of family-based problem-solving, the disruption of negative critical parent–child interaction, and the promotion of secure parent–child attachment. In a multi-country study comparing psychodynamic individual and family therapy interventions for children aged 9 to 15 years presenting with moderate to severe depression, both approaches were found to be effective [14]. Over 74% of children in both groups were no longer clinically depressed at post-test, and 81% of the family therapy group were also no longer clinically depressed at 6-month follow-up.

Attempted suicide

Family interventions have been found to improve the adjustment of adolescents who have attempted suicide [15], while a version of MST adapted for such young people was more effective than emergency hospitalization and treatment by a multidisciplinary psychiatric team [16]. Effective approaches begin with engaging young people and families in an initial risk-assessment process, then developing a clear plan for risk reduction involving individual therapy for adolescents alongside systemic therapy for members of the family and social support networks. King *et al*. [17] describe a manualized Youth-Nominated Support Team approach that involves the young person naming four people to be part of their 'support team'. This team, which might include individuals in schools, extended family or religious community, is encouraged to maintain weekly contact with the adolescent and themselves receive input aimed at facilitating their understanding of the young person and their provision of appropriate support. Compared with psychotherapy and antidepressant medication, this approach led to improvements in the level of suicidal ideation for girls, but not significantly for boys. The authors hypothesized that because female adolescents tend to perceive higher levels of social support than male adolescents and are usually more satisfied with the social support they receive from persons in their lives, the enhanced support system involved in the intervention would be particularly helpful for female subjects. Their research suggests that outside-the-family supports can be useful for suicidal adolescents because some parents of suicidal teenagers have significant difficulties of their own that interfere with their ability to be supportive.

EATING DISORDERS

Adolescent anorexia nervosa

Family therapy approaches here as described by Eisler [18] involve firstly an 'engagement' phase making contact with each adolescent's family member and emphasizing a primary task of overcoming anorexia, rather than understanding its causes; secondly helping the family to 'challenge the symptoms'; thirdly as concerns around eating recede, exploring issues of individual and family development more broadly; and fourthly ending with a discussion of future plans. Eisler notes also [18] the usefulness of a multiple family day programme where different families can meet and establish group cohesion in a supportive atmosphere in which new solutions can be tried.

Eisler's [18] systematic review of 11 family therapy trials for adolescent anorexia nervosa found

that by the end of treatment between one-half and two-thirds of participants had achieved a healthy weight. At follow-up between 60% and 90% had fully recovered. This contrasts with the rates of relapse of 25–30% for first in-patient admission and 55–75% for further admissions. This evidence is reflected in the National Institute for Health and Clinical Excellence (NICE) guidelines [19], which state that: 'Family interventions directly addressing the eating disorder should be offered to children and adolescents with anorexia nervosa'. Some caveats are noted by Eisler, however, including the small number of studies, their methodological limitations, and that there is little research comparing family therapy with other treatments. He notes, too, that systematic evaluations have largely been confined to family therapy with a strong 'structural' flavour [18].

Bulimia

Two trials of family therapy show it to be more effective than supportive therapy [20], and as effective as CBT interventions [21] that also help parents to work together to supervise the young person to break the binge–purge cycle.

A DIFFERENT SORT OF EVIDENCE

Narrative/postmodernist family therapists would argue that an exclusive reliance on knowledge drawn from RCTs of manualized approaches to treatment ignores the more 'local' knowledge and expertise in managing difficulties developed by clinicians, clients, families, services and communities. This can be disempowering and unhelpful to families. Fredman [22], in contrast, illustrates how clinicians' knowledge and expertise, combined with specific knowledge gained during the therapeutic encounter, can help bereaved families.

An alternative method of gaining 'practice-based evidence' has been described by Young and Cooper [23] whereby families who had received therapy reviewed tapes of their own clinical sessions. Families were asked to stop the tape at 'meaningful moments' and then interviewed. The following themes were generated:

- Family members commented upon the importance of their **therapist's stance** – e.g. a 14-year-old commented, '*I liked hearing that she was accepting that if I didn't want to answer a question then she would be fine with that*. . .'.
- **Giving people back their words**: an 11-year-old commented that '*with her reviewing the stuff I said, it just really helped me 'cause it was in my brain more*. . .'.
- **Externalizing conversations**: a mother of an 8-year-old said, '*What she was doing in terms of how she was phrasing things, because she said ''the worry puts thoughts in your head*. . .'' *and my son was immediately saying, like echoing back what she was saying, ''the worry does this*. . .'' *so I was starting to feel that this was looking good.*' [laughs].

Such action-based research can help ensure that clinicians are attentive to the experience of service-users and that their practice is responsive and effective. It also acts as a counterweight to knowledge derived from the research on trials of manualized treatment approaches that have formed the bulk of this chapter. This emphasis on what clinicians bring to their work and how they learn and develop is an important complement to lessons about effectiveness drawn from larger scale quantitative studies. It has long been found that the specific technique or approach used by therapists is not as important in accounting for effectiveness as non-specific factors linked to the quality of the relationship that is developed between client (family) and therapist (as evidenced e.g. by Chatoor and Krupnick's [24] review of the literature). The responsible and ethical systemic practitioner will be able to draw from the research knowledge base developed for the particular problem areas described above, and continue to learn, from their practice with individual clients, what contributes to the development of relationships that client families experience as helpful.

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Psychotherapeutic Approaches: A Psychodynamic Perspective

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INTRODUCTION

An interest in applying psychoanalytic ideas to therapeutic work with children and adolescents arose out of the thinking of Anna Freud and Melanie Klein, and received further impetus from work showing the impact upon children of their experiences of separation and loss during the Second World War [1]. Over time psychoanalytically informed therapeutic approaches have expanded beyond individual work with the child to include work with families, groups, parent–infant psychotherapy and parent/couple work. This chapter focuses on psychodynamic psychotherapeutic approaches to children and adolescents presenting with a variety of emotional and behavioural problems.

BASIC PREMISES OF A PSYCHODYNAMIC APPROACH

Key concepts guiding the therapeutic process include an interest in unconscious processes and the 'internal' world of the child. Children's play is thought to provide a window onto the child's unconscious thoughts and feelings. The child's play and behaviour is therefore used by the therapist to understand the child's inner world [2]. Attunement to the child in order to be receptive to the minutiae of what is being emotionally exchanged is one of the primary tasks [3]. The ability to gather fine details regarding how the child responds to the setting and relates to the therapist is essential.

Where possible the work is undertaken in a regular consistent setting. When working individually with young children the psychotherapist prepares a box for each child with suitable toys and drawing materials to facilitate the child's creative play, exploration and non-verbal communication. It is usual practice for the child's parents/carers to attend parallel psychotherapeutic sessions.

The psychotherapeutic model is deeply embedded in a developmental approach to children's difficulties, and an excellent account of a psychoanalytic perspective on personality development from infancy to adolescence can be found in Waddell [4]. Increasingly, research from neuroscience and developmental psychology is used to complement this model and enhance understanding of work with children with neurodevelopmental disorders or those who have experienced severe maltreatment [5].

THE EVIDENCE BASE FOR CHILD PSYCHOTHERAPY

The evidence base for psychodynamic child psychotherapy is somewhat limited as randomized controlled trials (RCTs) have been few in number. A 2004 systematic review identified 32 studies, noting that many had limitations of study design and sample size. Only five were RCTs and four were quasi-RCTs [6]. Though small in number, these latter nine studies represent 33.3% of the total reviewed – a proportion that compares favourably with the 7.4% of experimental/quasi-experimental

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studies identified in an examination of the evidence base for treatments in child mental health in general [7]. Recent research reviews include qualitative studies and those focusing on process as well as outcome research [8,9]. A National Institute for Health Research (NIHR) multicentre RCT, comparing cognitive–behavioural therapy, short-term psychodynamic psychotherapy and specialist clinical care, in the treatment of adolescents with major depression, is now underway (see the NIHR website: http://www.hta.ac.uk/project/1731.asp).

EXAMPLES OF RESEARCH WITH CHILDREN AND YOUNG PEOPLE PRESENTING WITH VARIOUS CLINICAL PROBLEMS

Children who have experienced abuse or neglect

While not concerned with assessing the effects of psychodynamic interventions per se, an interesting body of work undertaken by Hodges and Steele [10,11] illustrates that it is possible to assess and measure changes in the attachment representations of children who have been adopted following experiences of abuse and neglect. Using a story stem technique, whereby children are presented with the beginning of a story relevant to their experiences and then asked to complete it, Hodges and Steele demonstrated that the children's portrayal of attachment figures changed over a 2-year follow-up period to include more positive representations of attachment figures, although earlier negative representations still persisted alongside the more positive ones.

A research project by Trowell *et al*. [12], on the other hand, concentrates on assessing the effectiveness of two types of interventions for girls who had been sexually abused. Using an RCT design, individual psychotherapy was compared with a psycho-educational group psychotherapeutic intervention. While both types of intervention were found to be effective at substantially reducing psychopathological symptoms, and participants in both groups evidenced improved functioning, individual therapy led to a greater improvement in symptoms of post-traumatic stress disorder. The authors note that the small sample size, and the lack of a control group limit conclusions about changes attributable to treatment.

Internalizing and externalizing disorders

Disruptive behavioural disorders: There is limited intervention research in this area, though a retrospective study of the case notes of 763 children attending the Anna Freud Centre found poorer outcomes for those diagnosed with conduct or oppositional defiant disorders [13]. Outcomes were, however, better for younger children and those with mixed emotional and behavioural disorders.

Internalizing disorders: Research evidence here suggests that children with depressive and/or anxiety disorders respond positively to a psychodynamic therapeutic approach.

In a quasi-randomized 2-year follow-up of children aged 6–11 years with depressive or anxiety disorders, Muratori *et al*. [14] compared those assigned to either a time-limited psychodynamic psychotherapy (PP) intervention condition or to community services. The results of the study indicated that PP was effective in treating internalizing disorders at the time of intervention and at 6-month follow-up. A 'sleeper' effect for PP was also found at 2-year follow-up in so far as only children in the PP group moved into the non-clinical range on standardized assessments, while those in the control group remained at the same level of clinical severity.

A multicentre randomized trial compared focused individual psychodynamic therapy and parallel therapeutic work with parents (FIPP) with a systemic integrative family therapy approach (SIFT), in a sample of children aged 10–14 years who met criteria for major depressive disorder and/or dysthmia [15]. Significant reductions in disorder rates for both groups were found such that clinical depression had remitted in more than 70% of participants in both types of intervention, and reductions in comorbid conditions were evident. Improvements were persistent, with a 6-month follow-up indicating that none of the FIPP participants remained depressed, compared to 81% of SIFT participants, although the loss of four cases to follow-up in the SIFT group limited assessment of effectiveness rates. While the final outcome of these interventions appears similar, a different pattern of responses was found. Family work appeared to have highly effective initial impact, whereas the response to individual work was slower but possibly more sustained.

Mixed diagnoses

A number of studies have focused on children presenting in middle childhood with a range of difficulties rather than belonging to a particular diagnostic category. One randomized trial of such children, aged 5–9 years, compared time-unlimited or time-limited (12 sessions) psychodynamically oriented treatment with a minimal-contact control group (four sessions) [16]. All groups showed significant improvements from pre-test to post-test, though changes in family functioning in the control group were significantly greater than those in the time-unlimited group. At 4-year follow-up, all three groups did well on a variety of outcome measures although the control group did rather better, being the only group to report significant improvements on severity of target problems and measures of family functioning. The researchers speculate that the four-session 'minimal contact control' group may have proved most effective because the families' own capacities for coping and resilience had been harnessed.

A further RCT compared the effectiveness of structural family therapy with individual psychodynamic child psychotherapy and a 'recreational' control in boys aged 6–12 years presenting with mixed diagnoses [17]. Attrition was greatest in the control group (43%) and greater in the family therapy group compared with the individual therapy (16% vs 4%). Both family therapy and individual psychodynamic therapy were equally effective in reducing behavioural and emotional problems on a variety of outcome measures that included family systems and individual psychodynamic rating scales. Findings on measures of family functioning were mixed: the control group showed no significant change; the family therapy group improved; those receiving individual psychodynamic psychotherapy showed deterioration at 1-year follow-up. This finding may possibly be biased as an intention-to-treat analysis was not carried out despite variable drop-outs in the three groups, but it may also be attributable to the fact that the individual psychodynamic child therapy was undertaken in the absence of any parallel parent work, contrary to usual practice. The study underlines the importance of working with the wider family system in conjunction with individual work with the child.

Young people with poorly controlled diabetes

Moran and colleagues undertook a series of studies assessing the effectiveness of psychoanalytic psychotherapy for children with poorly controlled diabetes [18,19]. A quasi-randomized study compared two groups, each containing 11 diabetic children with unstable insulin-dependent diabetes. Those in the treatment group received intensive psychoanalytic psychotherapy (up to 3–4 times a week) for an average of 15 weeks; those in the control group received only routine psychological input without individual psychotherapy. A significant improvement in diabetic control was noted in the experimental group compared to controls, with 91% of participants in the treatment group showing a reduction in glycosylated haemoglobin in contrast to only 36% of controls. This improvement was maintained at 1-year follow-up.

As part of this study three children with diabetes and growth retardation were studied, using a single-case experimental design methodology; in all three cases there were gains in height over the predicted height following psychotherapeutic treatment [19].

Long-term outcomes

The Anna Freud Centre long-term follow-up study:

Adult outcome: In this study [20], the adult outcome of 34 children who had received psychotherapeutic treatment at the Anna Freud Centre was compared with the outcome of 11 of their untreated siblings. In general those who had received treatment in childhood were found to be functioning well, reporting low levels of adversity, relatively few severe life events and good health. They displayed adequate personality functioning across a range of domains and a low rate of personality disorders.

Interestingly while adversity in childhood was greater in the treated children, the untreated siblings were found to experience more negative life events in adulthood. In relation to personality functioning, the entire sample appeared to be doing well in the work domain. In the area of intimate relationships those children successfully treated in childhood appeared to be doing better than their untreated siblings.

Possible adverse effects of treatment were highlighted in relation to attachment security. While a secure adult attachment status was common in

those who had moved from poor functioning in childhood to high functioning in adulthood, the attachment style of those who had been *unsuccessfully* treated in their childhood was predominantly preoccupied/entangled. Those children in the sample who did not receive psychoanalytic treatment were found to be predominantly dismissing in their adult attachment style. Treated participants demonstrated a balanced and accurate memory of their childhood experiences, though in contrast to their siblings, their memories tended to be more painful.

Patients' perspectives: Another aspect of this study assessed the perspective of the patient by exploring the memories of adults who were in therapy as children and examining the meaning participants gave to the experience of therapy in the context of their later lives [21].

Two-thirds of participants were able to describe some aspect of the experience of child psychotherapy that had felt helpful at the time of treatment. Some were more confident about the positive impact than others. Several described how being able to talk and 'unburden' themselves was helpful. One described how the treatment provided a 'sort of canvas' to 'express myself in a way that I wouldn't necessarily have been able to talk to anyone else about these problems'. Some noted how the therapist's attention made them feel more confident and how therapy enabled them to cope better.

Others questioned the potentially negative impact of the therapy. Some of their comments included feelings that the therapy was 'pointless' and had made no difference, or that it had set them apart from others. As one observed 'the last thing I wanted was to feel different'. In some participants, this sense of being different created or exacerbated a sense that they were somehow 'damaged' and that there was 'something wrong' with them.

POTENTIAL ADVERSE EFFECTS OF TREATMENT

In contrast to research on pharmacological treatments there has been a tendency not to look systematically for adverse effects of psychotherapeutic treatments. However, existing research suggests some potential adverse consequences of treatment that would benefit from scrutiny in future research. For example, there are indications that individual psychotherapy undertaken in the absence of concurrent parent/family work may have a negative impact on family functioning. There is a suggestion also that unsuccessful treatment in childhood may result in a preoccupied/entangled attachment style in adulthood. In addition some adults who received treatment in childhood describe how the treatment itself compounded a sense they had that there was 'something wrong with them' and for some there was an anxiety that it may have resulted in a tendency to be overly introspective.

CONCLUSION

The application of psychoanalytic understanding to psychotherapeutic work with children has a long tradition. Hopefully this work will continue to evolve and develop with the contribution of new insights from large-scale treatment trials, developmental psychology and neuroscience.

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Paediatric Psychopharmacology: Special Considerations

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INTRODUCTION

Problems of mental health and behaviour in children require a multidisciplinary approach, and optimal treatment is multimodal. The number of children in the USA taking prescription drugs for emotional and behavioural disturbances is growing dramatically and has given rise to multiple controversies, ranging from concerns over off-label use and long-term safety to debates about the societal value and cultural meaning of pharmacological treatment of childhood behavioural and emotional disorders. More than 80% of the worldwide use of stimulant medications occurs in the USA, and the use of antidepressants and antipsychotics is many times greater in the USA than in other countries [1]. Variability in use reflects differences in diagnostic systems, clinical practice guidelines, drug regulation, health services organization, availability and allocation of financial resources, and cultural attitudes towards childhood behavioural and emotional disturbances [1]. This chapter focuses on aspects of psychopharmacology that have special relevance in children and adolescents; it provides relevant information about classes of medication, rather than disorder-specific treatment recommendations.

INFORMATION TO ASSIST JUDICIOUS PRESCRIBING

Apart from a thorough diagnostic assessment, the following information is important:

- full medical history – current and past;
- detailed medication history, including over-the-counter medications;
- history of substance misuse to ascertain potential misuse liability and interactions with prescribed medication;
- detailed family history, including history of mental illness, suicide, substance abuse, neurological/medical conditions (especially early-onset coronary artery disease), and the response of the family members to psychotropic medication.

MEDICATION AS PART OF A MULTIMODAL TREATMENT PACKAGE

Treatment plans should be individualized according to the pattern of target symptoms and strengths identified in the evaluation. Treatment should target situations in which symptoms cause most impairment, and treatment progress should be monitored by custom-designed target symptom scales or daily behavioural report cards. The designation of a case manager is essential for chronically disabled individuals to coordinate the wide range of services necessary for their care and to ensure periodic diagnostic reassessments.

SYMPTOM-BASED PHARMACOTHERAPEUTIC STRATEGY

As pharmacological treatment is symptom based in most psychiatric conditions, it is useful to conceptualize it as described below [2].

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- *Symptoms that require and are likely to respond to medication alone:* inattention, impulsivity, hyperactivity, tics, obsessions, psychotic symptoms, labile mood.
- *Symptoms that are less likely to respond to medication alone, requiring both medication and psychosocial interventions:* aggression, rituals, self-injury, depression.
- *Symptoms that are unlikely to respond to medication and need specific remediation:* skill deficits in academic, social or sports domain.

THE ART OF PRESCRIBING MEDICATION

Besides the neurochemical effect of any given agent, the response to medication also depends on an inherent 'placebo response', as well as the therapeutic concordance achieved by obtaining agreement and acceptance of why the medication is prescribed and what is the expected response. Rewards experienced from medication treatment include improvement in symptoms, school performance and family relationships, and reduced level of parenting stress. Identified costs include the impact of adverse side effects, social stigma, lack of response, fears of addiction, and changes in the child's personality [3]. Acceptance of the diagnosis influences adherence while medication education has varying effects. Families' attitudes, beliefs and perceptions about psychiatric illness and treatment play a large role in medication treatment decisions. A trusting relationship with the clinician has a positive effect on adherence, but psychosocial treatment alternatives are usually preferred. With maturation, adolescents have more influence on decisions related to adherence [3].

The above characteristics are enhanced when parents and patients feel understood, accept that treatment is necessary and agree with the prescriber regarding the need for the treatment, and when medication is started in small doses using the principles of minimum effective dose (MED). The MED is the minimum dose with which 'acceptable' improvement with minimal side effects is achieved. Medication should be initiated in small doses (usually in doses that are one-eighth to one-sixth of the final anticipated dose), increasing the dosage after about every five half-lives of the drug – in practice usually every 3–7 days, over a period of 4–6 weeks – to identify the MED [4].

USE OF NON-LICENSED PSYCHOTROPIC MEDICATION

Most psychotropics – other than stimulants, atomoxetine for attention-deficit hyperactivity disorder (ADHD), and imipramine for enuresis – are not licensed for use in children. Unlicensed psychotropics are not contraindicated in children, and doctors can prescribe any medication approved by the appropriate agency (e.g. the US Food and Drug Administration or the European Medicines Agency), to any age group, if they believe that there is a reasonable clinical indication. Thus, licensing of medication constrains drug companies but leaves doctors free to prescribe unlicensed drugs or to use licensed drugs for unlicensed indications. The drug companies are not legally liable if any untoward reaction occurs in children treated without their knowledge, using such non-licensed medication. It is therefore important that the parents and patients (as appropriate) are given this information as part of the informed consent.

FACTORS AFFECTING PHARMACOTHERAPY IN CHILDREN

Understanding the pharmacokinetics and pharmacodynamics of drugs used in psychopharmacology across the paediatric age spectrum from infants to adolescents represents a major challenge for clinicians. In paediatrics, treatment protocols use either standard dose reductions for these drugs for children below a certain age or use less conventional parameters such as weight for allometric dosing; the rationale behind this, however, is often lacking.

Absorption and hepatic metabolism: The rate of absorption is faster in children, and peak levels are reached sooner. Hepatic metabolism is highest during infancy and childhood (1–6 years), is about twice the adult rate in prepubertal children (6–10 years), and is equivalent to that in adults by the age of 15 years [5]. This is important clinically because younger children may require higher doses (mg/kg) of hepatically metabolized medication, compared with older children and adults.

Fat distribution: Substantial fat stores slow the elimination of highly lipid-soluble drugs (e.g. fluoxetine and pimozide) from the body. Fat distribution

varies in children, increasing during the first year and gradually falling until puberty.

Protein-binding and volume of distribution: These differ in children, affecting pharmacokinetics by modifying the fraction of drug that is active (unbound) [6].

Incomplete maturation of neurotransmitter system: The noradrenergic system does not fully develop anatomically and functionally until early childhood [7]. This may be one of the reasons for poor antidepressant response in childhood depression.

Cardiotoxicity: The rates of maturation of the sympathetic and parasympathetic system vary, although vagal and sympathetic modulations follow a similar pattern. This may lead to accentuation of the relative loss of vagal modulation associated with tricyclic antidepressants [8].

MEDICATIONS

The dose ranges of the majority of psychotropics used in children and adolescents, with their main indications, are shown in Table 46.1.

Stimulants

Stimulants have been used for decades and good research evidence exists for their short-term use in ADHD. More recently, various stimulant delivery systems have been developed – the osmotic controlled-release system (OROS), Concerta XL; the wax matrix-based beaded system, Metadate CD or Equasym XL; Focalin XL; the patch release system, Daytrana, etc. – resulting in long-acting preparations that make it possible to avoid the administration of medication in school, reducing stigmatization and embarrassment.

The release systems and preparation of stimulants (proportion of immediate release vs slow release) allows the tailoring of the long-acting preparations to suit individual children [9]. Stimulants are contraindicated in schizophrenia, hyperthyroidism, cardiac arrhythmias, angina pectoris and glaucoma, and in patients with a history of hypersensitivity. Stimulants can be used with caution in hypertension, depression, tics (or family history of Tourette syndrome), autism spectrum disorders, and severe mental retardation (Table 46.2).

Antipsychotics

Second-generation antipsychotics (SGAs): These are prescribed most frequently, and include risperidone, quetiapine, aripiprazole, olanzapine, ziprasidone and amisulpride. They are dopamine receptor blockers (hence they reduce positive symptoms but can produce extrapyramidal symptoms and hyperprolactinaemia) and 5HT-2A receptor blockers.

- *Risperidone* is the most used SGA; it is a potent dopamine D2 receptor blocker (hence produces hyperprolactinaemia) and can lead to extrapyramidal symptoms.
- *Quetiapine* is an effective SGA with a moderate effect on weight; it usually needs to be taken at least twice daily because of relatively weak receptor binding.
- *Ziprasidone* is the only SGA that is weight neutral; however, it has a greater impact on cardiac rhythm and the QTc interval.
- *Clozapine* is used in those with resistant psychoses or tardive dyskinesia, but can lead to neutropenia, sialorrhoea and significant weight gain.
- *Olanzapine* is used less in children and adolescents because of the propensity to weight gain and metabolic syndrome. Evidence from adults suggests that clozapine, olanzapine and low-potency conventional antipsychotics such as chlorpromazine are associated with an increased risk of insulin resistance, hyperglycaemia and type 2 diabetes mellitus.
- *Aripiprazole* is a dopamine partial agonist or dopamine stabilizer and also has actions at 5HT-2A and D3 receptors, and partial agonism of 5HT-1A receptors. Symptoms may improve in the first week, but it is recommended to wait 4–6 weeks to determine efficacy, owing to the pharmacokinetics of the drug. The mean elimination half-life of aripiprazole is 75 hours, and 94 hours for the major metabolite, dihydro-aripiprazole. Little published evidence exists on its use in managing non-psychotic disruptive behaviour in developmental disorders, although clinical experience suggests that very small doses (2–5 mg daily) are sufficient (Table 46.3).

Table 46.1 Dose range of psychotropic medication used in children and adolescents.

Drug	Dose range	Target symptoms
Stimulants		
Methylphenidate IR	5-60 mg/day	Inattention, hyperactivity, impulsivity, and behavioural problems related to ADHD
Concerta XL	18-72 mg/day	
Equasym XL	10-60 mg/day	
Medikinet Retard	10-60 mg/day	
Dexamfetamine	2.5-40 mg/day	
Non-stimulant		
Atomoxetine	1-1.2 mg/kg body weight/day	
Tricyclic antidepressants		
Imipramine, desipramine	<6 years: 10-20 mg/day	Bedwetting, hyperactivity, impulsivity, inattention
	>6 years: 10-75 mg/day	
Clomipramine	10-200 mg/day	Obsessions, compulsions
SSRIs		Depression (only fluoxetine approved), obsessions and compulsions (high doses may be needed), self-injurious behaviour, and anxiety-related aggression in autism spectrum disorder (low doses)
Fluoxetine	10-60 mg/day	
Fluvoxamine	50-300 mg/day	
Sertraline	25-150 mg/day	
Paroxetine	10-60 mg/day	
Citalopram	10-60 mg/day	
SNRI		
Venlafaxine	37.5-150 mg/day	Symptoms of ADHD in adults
Antipsychotic medication		High doses - psychosis, (hypo)mania
Haloperidol	Pre-pubertal: 0.5-8 mg/day	Low doses (<1/3 of dose for psychosis) - tics, severe aggression and self-injury; risperidone in ASD with ADHD
	Post-pubertal: 1-16 mg/day	
Clozapine*	50-600 mg/day	
Risperidone	0.25-6 mg/day	
Olanzapine	2.5-20 mg/day	
Quetiapine	25-300 mg/day	
Aripiprazole	1-15 mg/day	
Anti-epileptic medication		Epilepsy, symptoms and prophylaxis of bipolar illness
Carbamazepine	5-10 mg/litre (serum level)	
Sodium valproate	50-100 mg/litre (serum level)	
Clonazepam	0.5-4 mg/day	Epilepsy, akathisia, sleep disorders

(*continued overleaf*)

Table 46.1 *(continued)*

Drug	Dose range	Target symptoms
Other		
Lithium carbonate	0.4-1.0 mEq/litre (serum level)	Bipolar disorder, aggression in the learning disabled, augmentation in depression
Clonidine	0.05-0.4 mg/day	Hyperactivity, impulsivity, inattention, insomnia, tics, oppositionality, aggression in ASD
Buspirone	10-45 mg/day	Anxiety, hyperactivity, aggression
Melatonin	0.5-9 mg/day	Sleep problems
Naltrexone	12.5-50 mg/day	Severe resistant self-injurious behaviour in ASD

ADHD, attention-deficit hyperactivity disorder; ASD, autism spectrum disorder; SNRI, serotonin-norepinephrine reuptake inhibitor; SSRI, selective serotonin reuptake inhibitor.
*Only for psychosis and tardive dyskinesia.

Table 46.2 Specific side effects of stimulants.

Side effect	Precautions	Comment
Seizure	No evidence of decreasing seizure threshold; can be used in well-controlled epilepsy	If seizures appear or worsen, change to dexamfetamine; avoid atomoxetine
Growth retardation	Reduced height and weight centiles possible over time	Advisable not to start stimulants in children who are short or biologically predisposed to short stature
Cardiovascular problems	Increases heart rate and blood pressure - monitor regularly	High risk in those with structural cardiac defects; monitor with ECG. Avoid Adderall in cardiac high-risk groups
Abuse potential	Possible abuse of stimulants by others with access	Self-initiated increase in dose by emotionally unstable patients with substance use disorders is possible, and should be monitored; atomoxetine, bupropion or Concerta XL, (drug-delivery system makes it difficult to abuse) can be used
Psychotic symptoms	Can induce or worsen psychotic experience	Avoid in those who have first-degree relatives with a psychotic disorder or in children who have psychotic or quasi-psychotic experiences; atomoxetine, tricyclic antidepressants, clonidine, bupropion or risperidone can be used

ECG, electrocardiography.

Table 46.3 Specific side effects of selected psychotropic medication.

Drug	Precautions	Side effects
Non-stimulant		
Atomoxetine (noradrenaline reuptake inhibitor)	Contraindicated in hepatic impairment, glaucoma, uncontrolled seizures, or a history of hypersensitivity to drug; use with caution in hypertension, tachycardia, cardiovascular problems, and patients with long QT interval or family history of QT prolongation, or cerebrovascular disease	*Growth retardation:* reduction of two to three percentiles in mean height, and some weight loss *Seizure liability:* not to be used in patients with uncontrolled seizures, and should be discontinued in those who develop or have an increased frequency of seizures *Cardiovascular:* increases heart rate (by increasing noradrenergic tone) and small increase in blood pressure; QT interval prolongation *Suicide risk:* monitor for signs of depression, suicidal thoughts and behaviour* *Liver dysfunction:* severe liver injury—rare. Abnormal liver enzymes are more common. Discontinue on first symptom or sign of liver dysfunction, e.g. pruritus, dark urine, jaundice, right upper quadrant tenderness or unexplained flu-like symptoms
Tricyclic antidepressants		
Imipramine, amitriptyline, nortriptyline, desipramine, clomipramine	Use has declined due to concerns of cardiac arrhythmias and case reports of sudden death	Cardiotoxicity, danger of accidental or intentional overdose, troublesome sedation, anticholinergic side effects, lowered seizure threshold
Newer antidepressants		
SSRIS: fluoxetine, fluvoxamine, sertraline, paroxetine, citalopram, escitalopram *SNRI:* venlafaxine *NRI:* reboxetine, mirtazapine	It is currently advised that children or adolescents being started on, or dose being increased of, antidepressants should be monitored closely for emergence or worsening of suicidal ideation or behaviour	*Antidepressant-related suicidal ideation and behaviour:* consistently there has been increased suicidal ideation with use of antidepressants in childhood depression. This has to be balanced with genuine suicidal risk in untreated severe depression† *Antidepressant-induced behavioural activation:* increased motor activity, restlessness, excitability and impulsivity that occurs usually early in treatment and may be reduced by using the MED principle; managed by reducing the dose, and with a benzodiazepine for a few days

(*continued overleaf*)

Table 46.3 *(continued)*

Drug	Precautions	Side effects
Antipsychotic medication		
FGAs: haloperidol, chlorpromazine	Monitor movement disorders at baseline and regularly during treatment	*Extrapyramidal side effects* such as tardive dyskinesia are more common in FGAs. Aripiprazole and clozapine are useful in those who require antipsychotics but have developed tardive dyskinesia
SGAs: risperidone, olanzapine, quetiapine, aripiprazole, ziprasidone, clozapine	Monitor weight, waist circumference, and BMI at baseline and every 6 weeks; serum prolactin, fasting lipids, fasting cholesterol, fasting glucose, and liver function tests at baseline and every 6 months. Be cautious if there is a family history of obesity, dyslipidaemia, early-onset hypertension, cardiovascular disease, cerebrovascular accident or diabetes	*Hyperprolactinaemia:* common with risperidone and FGAs *Risk of weight gain and metabolic dysfunction:* High—clozapine, olanzapine Moderate—risperidone, quetiapine Low—amisulpride, aripiprazole, ziprasidone *Treatment of SGA-induced metabolic dysfunction:* preventive healthy lifestyle counselling; regular monitoring of body weight and metabolic variables; cognitive-behavioural therapy and motivational interviewing to address unhealthy diet, physical inactivity, and smoking; metformin therapy may become necessary in severe cases

FGA, first-generation antipsychotic; MED, minimum effective dose; NRI, norepinephrine reuptake inhibitor; SGA, second-generation antipsychotic; SNRI, serotonin-norepinephrine reuptake inhibitor; SSRI, selective serotonin reuptake inhibitor.

*A black box warning exists as a result of analyses showing more frequent suicidal ideation in clinical trials of children treated with atomoxetine [10].

†In December 2003, the Committee on Safety of Medicines concluded that the evidence was adequate to establish effectiveness only for fluoxetine in the treatment of depressive illness in children and adolescents, and advised against the use of the other SSRIs [11]. The US Food and Drug Administration has insisted on black box warnings for all SSRIs regarding the possibility of suicide-related behaviour as a side effect in children [12].

Mood stabilizers

Carbamazepine, sodium valproate, lamotrigine, lithium carbonate and SGAs are mood stabilizers.

- *Sodium valproate or valproic acid* is the most used mood stabilizer and is best avoided in girls of child-bearing age due to its teratogenic effects, as well as possible side effect of polycystic ovarian disease.
- *Lithium* use warrants regular blood level monitoring, which is often a problem in children.
- *Lamotrigine* is especially useful when significant depressive symptoms exist in bipolar disorder. Valproic acid markedly increases the half-life of lamotrigine and the likelihood of developing severe drug rashes including Stevens–Johnson syndrome. Lamotrigine is to be started at very low doses (as low as 5 mg/day) and increased slowly over a couple of months.

As antipsychotics, antidepressants and antimanic agents are more closely associated with the

development of obesity and sexual/reproductive adverse events in African American patients, practitioners need to carefully weigh the risks/benefits of prescribing psychotropic agents to African American children, taking into consideration pre-existing/comorbid conditions or individual risk factors for adverse reactions, especially when multiple medications are prescribed [13].

DRUG INTERACTIONS

Detailed reviews of the cytochrome P450 enzyme system in children and guidelines for the prediction of drug–drug interactions are available [14,15]. It is advisable to look through this list before prescribing concomitant medication. Terfenadine, ketoconazole, azetamazole and erythromycin, if co-administered with selective serotonin reuptake inhibitors, can in theory lead to cardiac arrhythmias. Fluvoxamine can significantly increase clozapine levels; sodium valproate significantly increases lamotrigine levels, and imipramine when added to erythromycin can lead to toxic delirium.

ETHICAL ISSUES IN PAEDIATRIC PSYCHOPHARMACOLOGY

Research on psychopharmacological treatment in children and adolescents is the subject of ongoing ethical discussion, as minors with mental disorders constitute a vulnerable patient group. Incentives for the conduct of clinical trials with children comparable to those contained in US legislation are now provided in the EU. Research to develop 'me-too' preparations (or drugs that are just similar to already existing drugs) may have no significant benefit for children, but can cause research burden and detract from clinically more important projects by utilizing limited investigator time and patient resources [16]. The issues of avoiding undue influence from funders and conflicts of interest remain a prominent concern that can be solved by declaring conflicts and publishing all results of studies extensively.

CONCLUSION

Pharmacogenetic studies may bring more individualized treatment approaches into child psychiatry but they remain at present a promise for the future. Please see Chapter 41. A holistic biopsychosocial formulation and management of the child's problem is essential as psychopharmacotherapy is only part of a package of care. Use of the MED principle assists in titrating initial dose increments to the expected target dose based on treatment response and emergent adverse effects. Paediatric pharmacovigilance for psychotropic agents and true long-term studies on efficacy and side effects are essential. Evidence on treatment impact on comorbid disorders, cost-effectiveness and impact on quality of life is sparse and urgently needs to be addressed. Until such detailed data become available, it is safe to assume that paediatric populations are at least as, or more, vulnerable to adverse effects as adults.

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